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NEBRASKA SWINE REPORT

- Nutrition
- Health
- Genetics
- Management

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Prepared by the staff in Animal Science and cooperating Departments for use in Extension, Teaching, and Research programs.

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Agricultural Research Division
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2009 Nebraska Swine Report

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Effect of Corn Dried Distillers Grains with Solubles (DDGS) on Growth Performance and Carcass Characteristics of Growing-Finishing Gilts with Previous Exposure to DDGS in the Nursery

The inclusion of high concentrations of DDGS (30%) in both the nursery and growing-finishing periods may result in negative effects on growth performance and carcass characteristics.

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Summary

The objective of this experiment was to evaluate the effects of high concentrations of distillers dried grains with solubles (DDGS; 30%) on growth performance and carcass characteristics of gilts, during growing-finishing, that were previously fed high concentrations of DDGS during the nursery phase. Overall (week 1 to 16), the following observations are noteworthy: 1) among pigs that were fed DDGS in the nursery, average daily gain (ADG) and final body weight (BW) tended ($P < 0.10$) to be lower during growing-finishing compared to pigs that did not receive DDGS in the nursery; 2) among pigs that received DDGS during growing-finishing, ADG tended ($P < 0.10$) to be lower compared to pigs that did not receive DDGS during growing-finishing; and 3) among pigs that received DDGS in both the nursery and during growing-finishing, ADG and final BW was decreased ($P < 0.04$) compared to pigs with no prior exposure to DDGS. With respect to carcass characteristics, 10th-rib back fat was greater ($P < 0.05$) at the end of finisher 2 among pigs that did not receive DDGS in the nursery and hot carcass weight tended ($P < 0.07$) to be decreased among pigs that received DDGS in both the nursery and during growing-finishing. This research indicates that the inclusion of high concentrations of DDGS in both the nursery and growing-finishing periods may result in negative effects on growth performance and carcass characteristics.

Introduction

Distillers dried grains with solubles (DDGS) is the primary co-product of ethanol production that is used in the pork industry. It has been estimated that approximately 15% of the DDGS that is produced is used in the pork industry, with the majority utilized in growing-finishing diets. Previous research with growing-finishing pigs has shown that the addition of DDGS up to 10% of the diet results in similar growth performance when compared to typical corn-soybean meal diets (Table 1). However, with the inclusion of DDGS in excess of 10%, growth performance may be compromised if diets are not formulated on a digestible amino acid basis. Less emphasis has been placed on utilization of DDGS during the nursery period and, to our knowledge, no experiments have been conducted to evaluate the growth performance of growing-finishing pigs that were exposed to high concentrations of DDGS during the nursery phase of production. The objective of this experiment was to evaluate the effects of high concentrations of DDGS (30%) on growth performance and carcass characteristics of gilts, during the growing-finishing phase, that were previously fed high concentrations of DDGS (30%) during the nursery phase.

(Continued on next page)

Table 1. Effect of dietary DDGS level on overall growth performance of growing-finishing pigs.[a]

	DDGS, %			
Item	0	10	20	30
ADG, lb	1.90[a]	1.90[a]	1.83[bc]	1.79[bd]
ADFI, lb	5.25	5.22	5.09	5.18
G:F, lb/lb	0.36[a]	0.36[a]	0.36[a]	0.34[b]
Final BW, lb	257.93[a]	258.93[a]	251.94[b]	246.94[b]

[a,b]Means within a row with unlike superscripts are different ($P < 0.05$)
[c,d]Means within a row with unlike superscripts are different ($P < 0.10$)
Shurson, J. 2006. 67th Minnesota Nutrition Conference, St. Paul, Minn.

Table 2. Composition of growing-finishing diets (as-fed basis) %.

	Grower 1 (week 1 to 3)		Grower 2 (week 4 to 8)		Finisher 1 (week 9 to 12)		Finisher 2 (week 13 to 16)	
	DDGS[a], %							
Item, %	0	30	0	30	0	30	0	30
Corn	69.2	55.8	73.1	58.5	78.7	63.8	84.7	64.1
Soybean meal, 47.5% CP	25.5	8.7	22.0	6.3	16.7	1.5	10.8	1.5
Tallow	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Dicalcium phosphate	1.3	0.7	0.9	0.4	0.7	0.1	0.7	0.0
Salt	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Limestone	0.8	1.3	0.8	1.1	0.8	1.3	0.8	1.2
Vitamin premix[b]	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Trace mineral mix[c]	0.2	0.2	0.2	0.2	0.1	0.1	0.1	0.1
L-Lysine•HCl	0.1	0.4	0.1	0.5	0.1	0.3	0.1	0.1
L-Tryptophan	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
L-Threonine	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
DDGS[c]	0.0	30.0	0.0	30.0	0.0	30.0	0.0	30.0
Analyzed Composition								
CP[d], %	16.83	16.08	16.19	15.12	14.12	13.57	12.13	13.79
EE[e], %	4.95	7.06	4.45	6.91	5.04	7.53	5.41	7.91
Calculated Composition								
Lysine, %	1.0	1.0	0.9	0.9	0.8	0.8	0.6	0.6
CP[d], %	18.0	18.0	16.6	16.6	14.5	15.2	12.2	15.0
ME[f], kcal/lb	1554	1625	1561	1478	1566	1639	1569	1645

[a]DDGS = Corn dried distillers grains with solubles
[b]Supplied per kilogram of diet at 0.2% inclusion: vitamin A supplied as retinyl acetate, 4,400 IU; cholecalciferol, 440 IU; a-tocopherol acetate, 24 IU; menadione sodium bisulfite, 3.5 mg; riboflavin, 8.8 mg; d-pantothenic acid, 17.6 mg; niacin, 26.4 mg; vitamin B_{12}, 26.4 mg
[c]Supplied per kilogram of diet at 0.1% inclusion: Zn (as ZnS_4O), 85 mg; Fe (as $FeSO_4•H_2O$), 85 mg; Mn (as (MnO), 20 mg; Cu (as $CuSO_4•5H_2O$), 7 mg; I (as $Ca(IO_3)•H_2O$, 0.17 mg; Se (as Na_2SeO_3), 0.17 mg
[d]CP = Crude Protein
[e]EE = Ether extract
[f]ME = Metabolizable energy

Materials and Methods

Animals

The experimental protocol was reviewed and approved by the Institutional Animal Care and Use Committee of the University of Nebraska–Lincoln. Twenty gilts [(Danbred × NE white line) × Danbred] were sorted by weight and randomly allotted to one of four dietary treatments in a 16-week experiment that was conducted at the University of Nebraska–Lincoln. Pigs (average initial BW 61.97 ± 1.6 lb) were individually housed in pens (6.3 × 3.4 ft) with wire flooring, one nipple waterer, and one stainless steel feeder under constant lighting in a temperature controlled room. Pigs had ad libitum access to feed and water. There were four treatments with one pig/pen and five replicates/treatment.

Treatments

Pigs utilized in the current experiment either had no previous exposure to DDGS or were previously exposed to 30% DDGS during phase 3 of the nursery period *(2008 Nebraska Swine Report)*. Among pigs that were fed 0% DDGS in the nursery, growing-finishing diets for the current experiment were formulated to provide either 0% DDGS (Treatment 1) or 30% DDGS (Treatment 2). Among pigs that were fed 30% DDGS in the nursery, growing-finishing diets for the current experiment were formulated to provide either 0% (Treatment 3) or 30% DDGS (Treatment 4). All diets were formulated on a total amino acid basis, fed in meal form and formulated to meet or exceed NRC requirements for growth (Table 2).

Data and Sample Collection

Pigs and feeders were weighed at the beginning of the experiment and biweekly thereafter. Feed disappearance was calculated using the difference between feed offered and feed remaining in the feeder at the end of each biweekly period. Body weight (BW) gain was calculated using the pig weight at the beginning and at the end of each biweekly period. Average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (G:F) were calculated based on the individual biweekly BW gain and feed disappearance. At the beginning of the experiment and at the end of Grower 1 (week 3), Grower 2 (week 8), Finisher 1 (week 12), and Finisher 2 (week 16), ultrasound was used to measure backfat thickness (BF) and longissimus muscle area (LMA) at the 10th rib. Carcass measurements (hot carcass weight, HCW; dressing percentage, DP; last-rib backfat, LRBF; 10th-rib BF; and LMA) were obtained at slaughter.

Statistical Analyses

Growth data were analyzed as a completely randomized design using the MIXED procedure of SAS. The main effect of the statistical models was dietary treatment. Pen was con-

Table 3. Body weights (BW), average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (G:F) of nursery pigs fed 0 or 30% DDGS without (Treatment 1 and 2) or with (Treatments 3 and 4) previous exposure to DDGS (30%) during the nursery period.

Treatment	1	2	3	4				
	DDGS[a], %							
Nursery	0	0	30	30				
Growing-Finishing	0	30	0	30				
						P-value		
Item					SEM[b]	1[c]	2[d]	3[e]
No. of pigs	5	5	5	5				
Initial BW, lb	63.41	62.99	59.49	61.98	1.61	0.12	0.57	0.29
Final BW, lb	270.2	250.6	248.2	235.6	9.70	**0.08**	0.11	**0.04**
Grower 1 (week 1 to 3)								
ADG[f], lb	1.56	1.45	1.70	1.48	0.18	0.66	0.37	0.93
ADFI[g], lb	3.95	3.70	3.75	3.75	0.35	0.73	0.56	0.39
G:F[h], lb/lb	0.40	0.39	0.45	0.39	0.01	0.44	0.34	0.70
Grower 2 (week 4 to 8)								
ADG, lb	2.40	2.27	1.92	2.12	0.13	**0.02**	0.84	**0.05**
ADFI, lb	5.36	4.74	4.34	5.11	0.35	0.13	0.71	**0.02**
G:F, lb/lb	0.45	0.49	0.44	0.43	0.01	0.15	0.90	0.84
Finisher 1 (week 9 to 12)								
ADG, lb	2.14	1.87	2.14	1.85	0.20	0.94	0.20	0.42
ADFI, lb	6.17	5.47	5.22	5.69	0.35	0.38	0.75	0.14
G:F, lb/lb	0.36	0.34	0.42	0.33	0.01	0.51	0.18	0.81
Finisher 2 (week 13 to 16)								
ADG, lb	2.05	1.83	1.85	1.50	0.18	0.14	0.13	0.13
ADFI, lb	6.15	6.22	5.60	5.60	0.35	0.25	0.95	0.55
G:F, lb/lb	0.34	0.30	0.32	0.25	0.01	0.30	**0.01**	**0.06**
Overall (week 1 to 16)								
ADG, lb	2.09	1.92	1.92	1.76	0.09	**0.10**	**0.08**	**0.04**
ADFI, lb	5.62	5.22	4.87	5.22	0.24	0.14	0.93	**0.08**
G:F, lb/lb	0.39	0.38	0.40	0.34	0.01	0.66	**0.01**	0.50

[a]DDGS = Corn dried distillers grains with solubles
[b]SEM = Standard error of the mean
[c]*P*-value: Orthogonal contrast to evaluate the effect of DDGS inclusion in the nursery [(1 + 2) vs. (3 + 4)]
[d]*P*-value: Orthogonal contrast to evaluate the effect of DDGS inclusion in growing-finishing [(1 + 3) vs. (2 + 4)]
[e]*P*-value: Orthogonal contrast to evaluate the effect of DDGS inclusion in the nursery [(1) vs. (2 + 3 + 4)]
[f]ADG = Average daily gain
[g]ADFI = Average daily feed intake
[h]G:F = Gain to feed ratio

sidered as the experimental unit and was considered as a random effect. In addition, orthogonal contrasts were utilized to evaluate the effect of previous inclusion of DDGS in the nursery (Treatments 1 and 2 vs. 3 and 4), to evaluate the effect of including DDGS during the growing-finishing period (Treatments 1 and 3 vs. 2 and 4), and to evaluate the effect of DDGS inclusion in both the nursery and during growing-finishing (Treatment 1 vs. 2, 3, and 4) on growth performance and carcass characteristics during the growing-finishing period.

Results and Discussion

Pig growth performance and BW results are summarized in Table 3. During Grower 1, growth performance was not affected by dietary treatment. During Grower 2, G:F was not affected by dietary treatment; however, ADG was decreased ($P < 0.02$) among pigs that received DDGS in the nursery (Treatments 3 and 4) compared to pigs that did not receive DDGS in the nursery (Treatments 1 and 2), and ADG and ADFI were decreased ($P < 0.05$ and 0.02, respectively for ADG and ADFI) in pigs that received DDGS (Treatments 2, 3, and 4) compared to pigs with no previous exposure to DDGS (Treatment 1).

During Finisher 1, growth performance was not affected by dietary treatment. During Finisher 2, ADG and ADFI were not affected by dietary treatment; however, G:F was greater ($P < 0.01$) for pigs that did not receive DDGS during growing-finishing (Treatments 1 and 3) compared to pigs that did receive DDGS during growing finishing (Treatments 2 and 4), and G:F tended ($P < 0.06$) to be greater for pigs with no prior exposure to DDGS (Treatment 1) compared to pigs that received DDGS during the nursery and growing-finishing (Treatments 2, 3, and 4). Overall, the following observations were made: 1) among pigs that were fed DDGS in the nursery, ADG and final BW tended ($P < 0.10$) to be lower during growing-finishing compared to pigs that did not receive DDGS in the nursery; 2) among pigs that received DDGS during growing-finishing, ADG tended ($P < 0.10$) to be lower compared to pigs that did not receive DDGS during growing-finishing; 3) among pigs that received DDGS in both the nursery and/or during growing-finishing, ADG and final BW was decreased ($P < 0.04$) and ADFI tended ($P < 0.08$) to be decreased compared to pigs with no prior exposure to DDGS; and 4) among pigs that did not receive DDGS during growing-finishing, G:F was greater ($P < 0.01$) compared to pigs that did receive DDGS during growing-finishing. Final BW were 270.2, 250.6, 248.2, and 235.6 lb, respectively, for Treatments 1, 2, 3 and 4.

Carcass characteristics are summarized in Table 4. Carcass measurements taken at slaughter (dressing percentage, last-rib BF, 10th-rib BF, and LMA) were not affected by dietary treatment; however, hot carcass weight tended ($P < 0.07$) to be decreased among pigs that received DDGS in both the nursery and during growing-finishing. Similar to final BW, live weight at slaughter tended ($P < 0.10$)

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to be decreased for growing-finishing pigs that received DDGS during the nursery period (Treatments 3 and 4) compared to pigs with no previous exposure to DDGS (Treatment 1 and 2). In addition, among pigs that received DDGS in both the nursery and/or during growing-finishing, live weight at slaughter was decreased ($P < 0.04$), compared to pigs with no prior exposure to DDGS. Ultrasound measurements taken at the end of Grower 1, Grower 2, Finisher 1, and Finisher 2 were not affected by dietary treatment with the exception of 10^{th}-rib BF. At the end of Finisher 2, 10^{th}-rib back fat was greater ($P < 0.05$) among pigs that did not receive DDGS in the nursery compared to pigs that received DDGS in the nursery.

Conclusions

This research indicates that feeding high concentrations of DDGS (30%) during the growing-finishing phase may not negatively affect growth performance. However, transient negative effects on overall ADG and final BW during the growing-finishing period may be observed in pigs that are fed high concentrations of DDGS in both the nursery and during growing-finishing.

[1]Thomas E. Burkey is an assistant professor, Phillip S. Miller is a professor, and Roman Moreno and Erin E. Carney are graduate students in the Animal Science Department at the University of Nebraska–Lincoln.

Table 4. Response and significance of dietary DDGS[a] inclusion on final weight and carcass characteristics of growing-finishing pigs without (Treatment 1 and 2) or with (Treatments 3 and 4) previous exposure to DDGS (30%) during the nursery period.

Treatment	1	2	3	4				
	DDGS[a], %							
Nursery	0	0	30	30				
Growing-Finishing	0	30	0	30				
						P-value		
Item					SEM[b]	1[c]	2[d]	3[e]
No. of pigs	5	5	5	5				
Live weight, lb	266.4	245.6	244.6	232.6	10.11	**0.10**	0.12	**0.04**
Carcass Measurements								
Hot carcass weight, lb	204.6	188.0	189.8	179.6	8.34	0.18	0.13	**0.07**
Dressing, %	76.81	76.56	77.51	77.18	0.75	0.39	0.71	0.75
Last rib BF[f], in	0.94	0.98	0.9	1.1	0.11	0.72	0.29	0.68
10^{th}-rib BF, in	0.86	0.84	0.74	0.76	0.07	0.17	0.99	0.33
LMA[g], in^2	11.07	10.6	11.13	10.51	0.54	0.98	0.33	0.61
Ultrasound Measurements								
10^{th}-rib BF, in								
Grower 1 (4 week)	0.42	0.39	0.41	0.4	0.02	0.83	0.28	0.37
Grower 2 (8 week)	0.47	0.51	0.45	0.49	0.02	0.33	0.11	0.70
Finisher 1 (12 week)	0.58	0.59	0.55	0.57	0.04	0.54	0.68	0.81
Finisher 2 (16 week)	0.79	0.78	0.64	0.71	0.05	**0.05**	0.55	0.20
10^{th}-rib LMA, in^2								
Grower 1 (4 week)	2.89	2.81	2.88	2.65	0.17	0.62	0.37	0.56
Grower 2 (8 week)	4.47	4.72	4.34	4.5	0.24	0.48	0.41	0.85
Finisher 1 (12 week)	5.47	5.1	5.41	5.24	0.2	0.83	0.19	0.35
Finisher 2 (16 week)	7.43	6.87	7.26	6.77	0.47	0.77	0.27	0.40

[a]DDGS = Corn dried distillers grains with solubles
[b]SEM = Standard error of the mean
[c]*P*-value: Orthogonal contrast to evaluate the effect of DDGS inclusion in the nursery [(1 + 2) vs. (3 + 4)]
[d]*P*-value: Orthogonal contrast to evaluate the effect of DDGS inclusion in growing-finishing [(1 + 3) vs. (2 + 4)]
[e]*P*-value: Orthogonal contrast to evaluate the effect of DDGS inclusion in the nursery [(1) vs. (2 + 3 + 4)]
[f]BF = Backfat
[g]LMA = Longissimus muscle area

Feeding Value of Diets for Growing-Finishing Pigs with Varying Concentrations of Corn Distillers Dried Grain with Solubles (DDGS)

Growth performance of growing-finishing pigs was maintained as dietary DDGS inclusion increased from 0 to 15%.

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Summary

Two-hundred and forty pigs (61.73 lb) were used in a 16-week study conducted to evaluate the feeding value of diets with varying concentrations of distillers dried grains with solubles (DDGS) for growing-finishing pigs. Pigs were assigned to one of four dietary treatments. Treatments consisted of a standard diet formulated on a standardized ileal digestible lysine (SID lys) basis in which a portion of dietary corn and soybean meal were replaced to include 0, 5, 10 or 15% of DDGS in a 4-phase feeding regime. Treatment did not affect average daily gain (ADG), average daily feed intake (ADFI) or gain/feed (G:F) during the Grower 1, Grower 2, Finisher 1, and Finisher 2 feeding periods (P > 0.10). Overall, no linear or quadratic effects in ADG and ADFI were recorded as dietary DDGS increased (P > 0.10). At day 21 and 42 backfat (BF) linearly decreased as dietary DDGS concentration increased (P = 0.008 and 0.018, respectively). A linear reduction in longissimus muscle area was recorded on day 42 (P = 0.025). Overall, growth performance was not affected by dietary DDGS inclusion increasing from 0 to 15%. The results of this study suggest that DDGS inclusion up to 15% in diets for growing-finishing pigs formulated on a SID lys basis does not affect optimum growth performance.

Introduction

The inclusion of dietary distillers dried grains with solubles (DDGS) in diets for growing finishing pigs represents a challenge from the diet formulation standpoint mostly due to the variation on nutrient composition among dietary DDGS sources imposed by the process by which starch is extracted. This variation in the composition of the DDGS is responsible in part for the variation in the growth performance of growing-finishing pigs fed diets in which DDGS has been included. Data reported in a previous study *(2008 Nebraska Swine Report)* using the same dietary DDGS inclusion showed that growth performance was linearly decreased as dietary DDGS increased form 0 to 15% . We attributed the inability of DDGS-supplemented diets to maintain maximum growth performance to the increased fiber concentration. In the present study, we screened the DDGS for lysine, crude protein, and fiber concentration in order to formulate the diets with the adequate concentration of nutrients to maximize growth performance. The objective of this study was to evaluate the feeding value of diets with inclusion rates of DDGS of 0, 5, 10 and 15% formulated in a standardized ileal digestible lysine (SID lys) basis for growing-finishing pigs.

Procedures

Animals and Facilities

This experimental protocol was reviewed and approved by the Institutional Animal Care and Use Committee of the University of Nebraska–Lincoln. For this 16-week study, 240 barrows and gilts [(Danbred × NE white line) × Danbred] were used. The initial average weight was 61.7 lb. Five barrows and five gilts were housed in each of 24 pens, and there were six replicates for each of the four dietary treatments.

Pigs were housed in a 24-pen building equipped with automatic environmental control. Pens dimensions were 4.95 × 15.84 ft and each pen was equipped with automatic feeder and waterer. The flooring was half solid concrete and half concrete slats. Pigs had ad libitum access to feed and water throughout the experimental period.

Dietary Treatments

The DDGS used for this experiment was analyzed for total lysine concentration and SID lys was calculated and used to formulate the experimental diets to ensure an adequate lysine supply to maximize growth performance. Pigs were fed diets that included 0, 5, 10 and 15% dietary DDGS formulated in a SID lys basis and arranged in a 4-phase dietary growing-finishing regime (Tables 1 and 2). Crystalline lysine was incorporated into diets containing DDGS

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Table 1. Ingredient, calculated and analyzed composition of growing diets, as-fed basis.

	Grower 1 (45 to 80 lb)				Grower 2 (80 to 130 lb)			
	DDGS[a], %							
Item, %	0	5	10	15	0	5	10	15
Corn	71.27	67.36	63.63	60.42	74.47	70.52	66.66	2.37
Soybean meal, 47.5 % CP[d]	23.75	22.75	21.5	19.75	21	20	19	18.25
Tallow	2	2	2	2	2	2	2	2
Dicalcium phosphate	1.2	1.12	1.05	0.95	0.85	0.75	0.65	0.6
Limestone	0.89	0.92	0.97	1.025	0.84	0.9	0.95	0.97
Salt	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Vitamin premix[b]	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Trace mineral mix[c]	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
L-Lysine•HCL	0.15	0.15	0.17	0.19	0.15	0.15	0.15	0.15
L-Tryptophan	0.01	0.01	0.01	0.01	0	0	0	0
L-Threonine	0.05	0.02	0.01	0	0.03	0.02	0	0
DL-Methionine	0.02	0	0	0	0	0	0	0
DDGS	0	5	10	15	0	5	10	15
Analyzed composition								
CP[d], %	16.61	17.18	17.521	8.23	15.66	16.36	16.60	17.25
EE[e], %	4.58	4.96	5.37	5.65	4.53	4.91	5.15	5.66
Calculated composition								
SID[f] lysine, %	0.9	0.9	0.9	0.9	0.83	0.83	0.83	0.83
CP[d], %	17.1	17.6	18	18.2	16.1	16.6	17	17.6
ME[g], kcal[h]/lb	1,543	1,530	1,517	1,505	1,550	1,537	1,525	1,512

[a]DDGS = Corn distillers dried grain with solubles
[b]Supplied per kilogram of diet at 0.2% inclusion: vitamin A supplied as retinyl acetate, 4,400 IU; cholecalciferol, 440 IU; a-tocopherol acetate, 24 IU; menadione sodium bisulfite, 3.5 mg; riboflavin, 8.8 mg; d-pantothenic acid, 17.6 mg; niacin, 26.4 mg; vitamin B_{12}, 26.4 mg
[c]Supplied per kilogram of diet at 0.15% of inclusion: Zn (as ZnS_4O), 128 mg; Fe (as $FeSO_4 \cdot H_2O$), 128 mg; Mn (as MnO), 30 mg; Cu (as $CuSO_4 \cdot 5\ H_2O$), 10.5 mg; I (as $Ca(IO_3) \cdot H_2O$), 0.26 mg; Se (as Na_2SeO_3), 0.26 mg
[d]CP = Crude protein
[e]EE = Ether extract
[f]SID = Standardized ileal digestible
[g]ME = Metabolizable energy
[h]Kcal = Kilocalories (1,000 cal)

Table 2. Ingredient, calculated and analyzed composition of finishing diets, as-fed basis.

	Finisher 1 (130 to 190 lb)				Finisher 2 (190 to 250 lb)			
	DDGS[a], %							
Item	0	5	10	15	0	5	10	15
Corn	80.18	76.03	71.83	67.85	86.58	82.3	78.56	74.25
Soybean meal, 47.5 % CP[d]	15.5	14.75	14	139.25	8.6	7.4	6.75	
Tallow	2	2	2	2	2	2	2	2
Dicalcium phosphate	0.7	0.65	0.56	0.47	0.6	0.5	0.42	0.35
Limestone	0.84	0.87	0.9	0.97	0.82	0.87	0.91	0.95
Salt	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Vitamin premix[b]	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Trace mineral mix[c]	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
L-Lysine•HCL	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
L-Tryptophan	0	0	0	0	0.01	0	0	0
L-Threonine	0.07	0	0	0	0.04	0.02	0	0
DL-Methionine	0	0	0	0	0	0	0	0
DDGS[a]	0	5	10	15	0	5	10	15
Analyzed Composition								
CP[d], %	13.00	14.01	14.42	15.14	10.83	11.67	12.32	13.06
EE[e], %	4.39	4.76	4.83	5.35	4.45	4.89	5.32	5.89
Calculated Composition								
SID[f] lysine, %	0.7	0.7	0.7	0.7	0.54	0.54	0.54	0.54
CP, %	14	14.6	15.2	16.3	11.6	12.2	12.4	13.3
ME[g], kcal[h]/lb	1,554	1,542	1,530	1,516	1,557	1,545	1,533	1,520

[a]DDGS = Corn distillers dried grain with solubles
[b]Supplied per kilogram of diet at 0.15% inclusion: vitamin A supplied as retinyl acetate, 3,300 IU; cholecalciferol, 330 IU; a-tocopherol acetate, 18 IU; menadione sodium bisulfite, 2.64 mg; riboflavin, 6.60 mg; d-pantothenic acid, 13.23 mg; niacin, 19.80 mg; vitamin B_{12}, 19.80 mg
[c]Supplied per kilogram of diet at 0.1% of inclusion: Zn (as ZnS_4O), 85 mg; Fe (as $FeSO_4 \cdot H_2O$), 85 mg; Mn (as MnO), 20 mg; Cu (as $CuSO_4 \cdot 5\ H_2O$), 7 mg; I (as $Ca(IO_3) \cdot H_2O$), 0.17 mg; Se (as Na_2SeO_3), 0.17 mg
[d]CP = Crude protein
[e]EE = Ether extract
[f]SID = Standardized ileal digestible
[g]ME = Metabolizable energy
[h]Kcal = Kilocalories (1,000 cal)

Table 3. Response and effect of dietary DDGS[a] inclusion on growth performance of growing-finishing pigs.

	DDGS[a], %					*P*-value		
Item	0	5	10	15	SEM[b]	Treatment	Linear	Quadratic
No. of pigs	60	60	60	60				
Grower 1 (day 0 to 21)								
BW[c] (day 0), lb	61.58	61.54	61.83	61.99	0.320	0.727	0.300	0.755
BF[d] (day 0), in	0.26	0.24	0.25	0.25	0.005	0.195	0.166	0.209
LMA[e] (day 0), in^2	1.65	1.66	1.64	1.62	0.028	0.793	0.368	0.676
ADG[f], lb	1.69	1.69	1.69	1.71	0.093	0.736	0.876	0.495
ADFI[g], lb	3.46	3.37	3.38	3.39	0.057	0.690	0.448	0.395
G:F[h], lb/lb	0.49	0.50	0.46	0.50	0.024	0.657	0.931	0.595
BW (day 21), lb	97.06	98.53	97.45	97.82	2.079	0.974	0.915	0.792
BF (day 21), in	0.38	0.37	0.34	0.35	0.011	0.030	0.008	0.352
LMA (day 21), in^2	2.79	2.84	2.71	2.66	0.046	0.060	0.025	0.283
Grower 2 (day 21 to 42)								
ADG, lb	1.79	1.66	1.80	1.73	0.108	0.791	0.901	0.768
ADFI, lb	4.36	4.29	4.26	4.39	0.170	0.931	0.932	0.533
G:F, lb/lb	0.41	0.39	0.43	0.39	0.017	0.395	0.868	0.680
BW (day 42), lb	134.77	133.42	132.53	134.60	2.291	0.965	0.996	0.674
BF (day 42), in	0.38	0.34	0.36	0.34	0.009	0.032	0.018	0.412
LMA (day 42), in^2	3.57	4.46	3.52	3.50	0.060	0.156	0.205	0.258
Finisher 1 (day 43 to 70)								
ADG, lb	1.85	1.96	1.88	1.94	0.077	0.735	0.555	0.774
ADFI, lb	5.83	5.69	5.81	5.85	0.201	0.948	0.851	0.676
G:F, lb/lb	0.32	0.35	0.33	0.33	0.009	0.192	0.524	0.278
BW (day 70), lb	190.66	191.08	189.42	188.97	3.369	0.830	0.659	0.897
BF (day 70), in	0.55	0.50	0.50	0.50	0.020	0.188	0113	0.159
LMA (day 0), in^2	4.63	4.77	4.67	4.42	0.089	0.064	0.076	0.037
Finisher 2 (day 71 to 112)								
ADG, lb	1.79	1.67	1.71	1.76	0.064	0.545	0.892	0.191
ADFI, lb	6.74	6.32	6.45	6.51	0.183	0.450	0.502	0.203
G:F, lb/lb	0.27	0.26	0.27	0.27	0.007	0.921	0.748	0.546
BW (day 112), lb	268.02	262.90	263.70	262.90	4.536	0.830	0.481	0.639
BF (day 112), in	0.81	0.81	0.73	0.75	0.046	0.469	0.201	0.843
LMA (day 112), in^2	7.06	6.60	6.61	6.59	0.136	0.998	0.989	0.878
Overall (day 0 to 112)								
ADG, lb	1.78	1.76	1.74	1.79	0.053	0.888	0.994	0.467
ADFI, lb	5.39	5.25	5.24	5.36	0.123	0.791	0.840	0.332
G:F, lb/lb	0.33	0.33	0.33	0.33	0.004	0.892	0.688	0.910
Carcass characteristics								
Hot carcass weight, lb	208.38	207.56	204.92	203.46	4.01	0.807	0.344	0.938
DP[i], %	74.20	74.90	73.80	74.40	0.04	0.316	0.751	0.867

[a]DDGS = Corn distillers dried grain with solubles
[b]SEM= Standard error of the mean
[c]BW = Body weight
[d]BF = Backfat
[e]LMA = Longissimus muscle area
[f]ADG = Average daily gain
[g]ADFI = Average daily feed intake
[h]G:F = Gain to feed ratio
[i]DP = Dressing percentage. DP = (live weight/hot carcass weight) × 100

in order to maintain a constant SID lys concentration among diets within feeding phases. Other nutrient concentrations were formulated to meet or exceed allowances identified in the Nebraska–South Dakota Swine Nutrition Guide.

Data and Sample Collection

Pigs and feeders were weighed at the beginning of the experiment and at the end of each dietary phase. Feed disappearance was estimated by the difference between feed offered and feed remaining in the feeder at the end of each feeding phase. Body weight gain was estimated by the difference between the weight at the beginning and at the end of each feeding phase. Average daily gain (ADG), average daily feed intake (ADFI) and ADG:ADFI (G:F) were estimated based on the individual body weight gain at the end of each feeding phase and feed disappearance. At the beginning of the experiment and at the end of each feeding phase, ultrasound was used to measure backfat thickness (BF) and longissimus muscle area (LMA) at the 10^{th}-rib area. At the end of the feeding phase pigs were transported to a commercial facility and harvested.

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Live weight and hot carcass weight (HCW) were recorded and dressing percentage was calculated [DP; DP = (live weight/HCW) × 100].

Statistical Analysis

The MIXED procedure (SAS Inst. Inc. Cary, N.C.) was used to analyze the data. Contrasts were designed to evaluate linear and quadratic responses to addition of DDGS to dietary treatments. Pen was considered the experimental unit, and the model was a completely randomized design. Pen was considered a random effect.

Results and Discussion

The growth performance responses of growing-finishing pigs to varying dietary concentrations of DDGS are provided in Table 3. During the Grower 1 period (day 0 to 21), treatments did not affect ADG, ADFI, or G:F ($P > 0.05$); similarly, no linear or quadratic responses to dietary DDGS inclusion were detected by the examination of contrasts ($P > 0.05$). There was a linear reduction on BF in response to dietary DDGS inclusion ($P = 0.008$), with the greatest BF (0.38 in) recorded for pigs fed diets with 0% dietary DDGS inclusion while the lowest BF (0.34 in) was recorded by the pigs fed 10% dietary DDGS inclusion. Although there was only a trend for a treatment effect on LMA ($P = 0.06$) the examination of the data indicated a linear reduction in LMA ($P = 0.025$) in response to dietary DDGS inclusion. The data showed that the smallest LMA (2.66 in^2) was for pigs fed diets with 15% DDGS inclusion, while the greatest LMA was for pigs fed diets with 0% DDGS inclusion (2.79 in^2).

Treatment did not affect ADG, ADFI, G:F, BW or LMA during the Grower 2 period ($P > 0.10$). A linear ($P = 0.018$) response of BF to dietary DDGS concentration indicated that BF decreased as dietary DDGS inclusion increased. The least BF (0.34 in) was for pigs fed 15% dietary DDGS and the greatest corresponded to pigs fed 0% dietary DDGS (0.38 in).

During the Finisher 1 period (day 43 to 70) no differences in ADG, ADFI, G:F, BW or BF were recorded ($P > 0.10$). There was a trend of LMA to decrease linearly in response to increased dietary DDGS concentration ($P = 0.076$).

During the Finisher 2 phase (day 71 to 112) there were no difference in ADG, ADFI, G:F, BF or LMA among treatments ($P > 0.10$). During this feeding phase the greatest ADG (1.79 lb) and ADFI (6.74 lb) was exhibited by the treatment formulated to have 0% dietary DDGS concentration. We showed no effect of treatment on BF and LMA at the end of the Finisher 2 feeding phase ($P = 0.469$ and 0.998 respectively); however, numerically the least BF (0.73 in) corresponded to pigs fed 10% dietary DDGS. Also LMA (7.06 in^2) was numerically the greatest for pigs fed 0% dietary DDGS inclusion. The final BW (day 112) data indicate no difference among treatments ($P = 0.830$).

For the overall period (day 0 to 112), our data indicate there was no difference among treatments on ADG, ADFI and G:F ($P = 0.888$, 0.791, and 0.892, respectively). There was no difference among treatments for HCW or DP ($P = 0.807$ and 0.316 respectively).

These data are in contrast to the results of our previous study *(2008 Nebraska Swine Report)* in which we found that increasing dietary concentration of DDGS from 0 to 15% resulted in a linear reduction in growth performance examined by ADG, ADFI and G:F. The reduced growth performance was partially attributed to increased concentration of neutral detergent fiber (NDF) in the experimental diets associated with the inclusion of DDGS. The results of the present experiment support the findings reported in the literature that indicate that DDGS may be included in diets of growing-finishing pigs up to 20% without negatively affect growth performance. The results of our experiment support the importance of screening DDGS samples for nutrient components especially CP, lysine, fat, and fiber.

Conclusions

Overall, growth performance of growing finishing pigs was maintained as dietary DDGS inclusion increased from 0 to 15%. This result may indicate that DDGS can provide lysine and other nutrients in adequate concentrations to maximize growth performance in growing-finishing pigs from the University of Nebraska herd.

[1]Roman Moreno is a graduate student and research technologist; Phillip S. Miller is a professor; and Thomas E. Burkey is an assistant professor in the Animal Science Department; Matthew W. Anderson is the manager; Jeffrey M. Perkins, Thomas E. McGargill, and Donald R. McClure are research technicians at the UNL Swine Research Farm.

Effects of Distillers Dried Grains with Solubles (DDGS) and Paylean® Supplementation on Growth Performance of Growing-finishing Pigs

Growth performance of growing-finishing pigs was not affected by increasing dietary DDGS (0 to 40%) or supplementing ractopamine.

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Summary and Implications

Forty pigs were used in a 14-week, 4-phase regime study conducted to evaluate the feeding value of diets with varying concentrations of DDGS for growing-finishing pig formulated on a standardized ileal digestibility (SID) lysine (lys) basis, DDGS withdrawal at the last feeding phase, and ractopamine (RAC)supplementation four weeks prior harvesting. Treatments consisted in 0, 15 or 40% dietary DDGS inclusion supplemented or not with RAC (4.5 ppm) four weeks prior harvesting. Increased dietary DDGS inclusion resulted in a linear reduction in average daily gain (ADG) during the Grower 1 period (P = 0.002). There were no treatment effects (P > 0.05) of increasing dietary DDGS inclusion for any of the variables examined during the Grower 2 feeding period. No differences among treatments were detected throughout the feeding phase Finisher 1 for ADG, average daily feed intake (ADFI), longissimus muscle area (LMA) and gain:feed (G:F) (P > 0.05). During the Finisher 2 feeding phase, there were no differences among treatments due to dietary DDGS inclusion on any of the variables studied. The inclusion of RAC four weeks prior harvesting did not affect growth performance (ADG, ADFI, and G:F; P = 0.436, 0.217, 0.880 respectively); however, there was a numerical increase in ADG due to RAC inclusion. The examination of 98-day BF and LMA data did not show differences due to RAC inclusion (P = 0.319 and 0.728 respectively). There were no changes in growth performance or ultrasound measurements due to withdrawal of DDGS (P > 0.05). Overall, growth performance was maintained as dietary DDGS inclusion increased from 0 to 40%.

Introduction

Despite the great quantity of information available in reference to nutrient composition and nutrient availability from DDGS, there is no consensus on the dietary inclusion that will maximize growth performance. Evidence available in the literature indicates that dietary inclusion levels up to 30% have been used in diets for growing-finishing without negatively affecting growth performance; however, the maximum amount of DDGS that can be included in the diet of growing-finishing pigs is still unclear.

The concentration of crude protein (CP) and lysine (lys) in DDGS is greater than that of corn; however, variability among sources has been reported. The inclusion of the beta-agonist ractopamine (RAC; Paylean®), has been shown to improve growth performance of finishing pigs when fed four weeks prior to harvesting. Additionally, RAC inclusion has resulted in increased average daily gain (ADG) and gain:feed (G:F), decreased carcass fatness, and increased carcass protein concentration; however, in order to produce these changes in growth performance and composition pigs fed RAC-supplemented diets require greater concentration of dietary AA (specially lys). The increased concentration of AA in DDGS makes it a viable option to use in conjunction with RAC supplementation. In addition, because dietary supplementation of DDGS has been associated with increased unsaturated fat content, dietary RAC addition, DDGS withdrawal during late finishing, or both may alleviate the problems with increased unsaturated fat content associated with DDGS feeding. Therefore, this study was conducted to examine the feeding value of diets with dietary DDGS concentrations of 15 and 40% formulated on a standardized ileal digestible (SID) lys basis and its interaction with the inclusion of RAC, DDGS withdrawal, or both during the last 4 weeks of the finishing period.

Procedures

Animals and Facilities

This experimental protocol was reviewed and approved by the Institutional Animal Care and Use Committee of the University of Nebraska–Lincoln. Forty barrows [(Danbred × NE white line) × Danbred] were used for this 14-week study. The initial average weight was 66.6 lb and the average final weight was 273.2 lb. Pigs were individually penned in fully-slotted pens equipped with automatic feeder and waterers to provide unlimited access to feed and water throughout the duration of the experimental period. Pigs were housed in a building equipped with automatic environmental control located in the UNL Swine Research Unit in Mead, Neb.

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Table 1. Ingredient, calculated and analyzed composition of growing diets, as-fed basis.

	Grower 1 (45 to 80 lb)			Grower 2 (80 to 130 lb)			Finisher 1 (130 to 190 lb)		
	DDGS[c], %								
	0	15	40	0	15	40	0	15	40
Treatment	T1	T2	T3 and T4	T1	T2	T3 and T4	T1	T2	T3 and T4
Item, %									
Corn	71.25	60.42	46.06	74.47	62.37	47.05	80.18	67.85	49.9
Soybean meal, 47.5% CP	23.75	19.75	9	21	18.25	8.5	15.5	13	6
Tallow	2	2	2	2	2	2	2	2	2
Dicalcium phosphate	1.2	0.95	0.6	0.85	0.6	0.25	0.7	0.47	0.1
Limestone	0.89	1.02	1.27	0.84	0.97	1.22	0.84	0.97	1.2
Salt	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Vitamin premix[a]	0.2	0.2	0.2	0.2	0.2	0.2	0.15	0.15	0.15
Trace mineral mix[b]	0.15	0.15	0.15	0.15	0.15	0.15	0.1	0.1	0.1
L-lysine•HCl	0.15	0.19	0.4	0.15	0.15	0.32	0.15	0.15	0.25
L-tryptophan	0.01	0.01	0.01	0	0	0	0	0	0
L-threonine	0.05	0	0	0.03	0	0	0.07	0	0
DL-methionine	0.02	0	0	0	0	0	0	0	0
DDGS[c]	0	15	40	0	15	40	0	15	40
Analyzed Composition									
CP[d], %	16.65	17.90	19.32	15.78	17.52	18.69	13.69	15.44	17.77
EE[e], %	4.78	5.80	7.70	4.83	5.87	7.99	5.10	5.92	7.92
Calculated Composition									
SID[f] Lysine, %	0.9	0.9	0.9	0.83	0.83	0.83	0.7	0.7	0.7
CP[d], %	17.1	18.2	18.6	16.1	17.6	18.3	14	15.6	17.3
ME[g], kcal[h]/lb	1,543	1,505	1,437	1,550	1,512	1,444	1,554	1,516	1,450

[a]Supplied per kilogram of diet at 0.2% inclusion: vitamin A supplied as retinyl acetate, 4,400 IU; cholecalciferol, 440 IU; a-tocopherol acetate, 24 IU; menadione sodium bisulfite, 3.5 mg; riboflavin, 8.8 mg; d-pantothenic acid, 17.6 mg; niacin, 26.4 mg; vitamin B_{12}, 26.4 mg. Supplied per kilogram of diet at 0.15% inclusion: vitamin A supplied as retinyl acetate, 3,300 IU; cholecalciferol, 330 IU; a-tocopherol acetate, 18 IU; menadione sodium bisulfite, 2.64 mg; riboflavin, 6.60 mg; d-pantothenic acid, 13.23 mg; niacin, 19.80 mg; vitamin B_{12}, 19.80 mg

[b]Supplied per kilogram of diet at 0.15% of inclusion: Zn (as ZnS_4O), 128 mg; Fe (as $FeSO_4$•H_2O), 128 mg; Mn (as MnO), 30 mg; Cu (as $CuSO_4$•5 H_2O), 10.5 mg; I (as $Ca(IO_3)$•H_2O), 0.26 mg; Se (as Na_2SeO_3), 0.26 mg. Supplied per kilogram of diet at 0.1% of inclusion: Zn (as ZnS_4O), 85 mg; Fe (as $FeSO_4$•H_2O), 85 mg; Mn (as MnO), 20 mg; Cu (as $CuSO_4$•5 H_2O), 7 mg; I (as $Ca(IO_3)$•H_2O), 0.17 mg; Se (as Na_2SeO_3), 0.17 mg

[c]DDGS = Corn distillers dried grain with solubles

[d]CP = Crude protein

[e]EE = Ether extract

[f]SID = Standardized ileal digestiblility

[g]ME = Metabolizable energy

[h]Kcal = Kilocalories (1,000 cal)

Dietary Treatments

The DDGS used for this experiment was analyzed for total lys concentration and this value was used to formulate diets and ensure an adequate lys supply to maximize growth performance. Diets were formulated on a SID basis arranged in a 4-phase dietary growing-finishing regime (Tables 1 and 2). Four dietary regimens were designed to provide DDGS inclusion of 0, 15 or 40% throughout the experiment or 40% dietary DDGS inclusion during the first three feeding phases and 0% dietary DDGS inclusion during the last feeding phase. Eight treatments were produced by randomly assigning pigs to 1 of 4 dietary treatments or their RAC- supplemented counterparts (4.5 ppm). Crystalline lys was incorporated in order to maintain a constant SID lys concentration among diets. Other nutrient concentrations were formulated to meet or exceed allowances identified in the Nebraska–South Dakota Swine Nutrition Guide.

Data and Sample Collection

Pigs and feeders were weighed and ultrasound was used to measure backfat thickness (BF) and longissimus muscle area (LMA) at the 10th rib at the beginning and at the end of each of four feeding phases. Feed disappearance was estimated by the difference between feed offered and feed remaining in the feeder at the end of each feeding phase. Body weight gain was estimated by the difference between the weight at the beginning and at the end of each feeding phase. Average daily gain (ADG), average daily feed intake (ADFI) and ADG:ADFI (G:F) were estimated based on individual body weight gain and feed disappearance during each feeding phase.

Statistical Analysis

Pen was considered a random effect and each pig was considered an experimental unit. Data were analyzed as a completely randomized design using repeated measures in time by the MIXED procedure (SAS Inst. Inc. Cary, N.C.). Contrasts were designed to evaluate linear and quadratic responses to dietary DDGS inclusion for the four feeding phases and overall. For the analysis of the data generated during the last feeding phase of the experimental period, contrasts were used to examine the effect of DDGS withdrawal and RAC inclusion.

Table 2. Ingredient, calculated and analyzed composition of finishing diets, as-fed basis

	Finisher 2 (130 to 190 lb)							
	DDGS[c], %							
	0	15	40	0	0	15	40	0
Item	T1	T2	T3	T4	T5	T6	T7	T8
Corn	86.56	74.97	55.1	86.56	72.23	62.36	45.98	72.23
Soybean meal, 47.5% CP	9.25	6	1	9.25	23.23	18.2	9.72	3.23
Tallow	2	2	2	2	2	2	2	2
Dicalcium phosphate	0.6	0.35	0	0.6	0.55	0.3	0	0.55
Limestone	0.82	0.95	1.15	0.82	0.61	0.77	0.97	0.61
Salt	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Vitamin premix[a]	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Trace mineral mix[b]	0.1	0.1	0.1	0.1	0.1	0.1	0.01	0.1
L-Lysine•HCl	0.15	0.17	0.2	0.15	0.46	0.54	0.67	0.46
L-tryptophan	0.01	0	0	0.01	0.04	0.04	0.04	0.04
L-threonine	0.05	0	0	0.05	0.15	0.11	0.05	0.15
DL-methionine	0	0	0	0	0.15	0.09	0	0.15
DDGS[c]	0	15	40	0	0	15	40	0
Paylean® (Ractopamine•HCL)	0	0	0	0	0.02	0.02	0.02	0.02
Analyzed Composition								
CP[d], %	10.79	12.80	16.01	10.84	17.20	18.20	20.03	17.50
EE[e], %	4.73	6.25	7.95	4.95	4.73	5.69	7.86	4.87
Calculated Composition								
SID[f] Lysine, %	0.54	0.54	0.54	0.54	1.13	1.13	1.13	1.13
CP[d], %	11.6	13	15.4	11.6	18.2	18.6	19.1	18.2
ME[g], kcal[h]/lb	1,557	1,519	1,454	1,557	1,505	1,437	1,446	1,505

[a]Supplied per kilogram of diet at 0.15% inclusion: vitamin A supplied as retinyl acetate, 3,300 IU; cholecalciferol, 330 IU; a-tocopherol acetate, 18 IU; menadione sodium bisulfite, 2.64 mg; riboflavin, 6.60 mg; d-pantothenic acid, 13.23 mg; niacin, 19.80 mg; vitamin B_{12}, 19.80 mg
[b]Supplied per kilogram of diet at 0.1% of inclusion: Zn (as ZnS_4O), 85 mg; Fe (as $FeSO_4 \cdot H_2O$), 85 mg; Mn (as MnO), 20 mg; Cu (as $CuSO_4 \cdot 5\ H_2O$), 7 mg; I (as $Ca(IO_3) \cdot H_2O$), 0.17 mg; Se (as Na_2SeO_3), 0.17 mg
[c]DDGS = Corn distillers dried grain with solubles
[d]CP = Crude protein
[e]EE = Ether extract
[f]SID = Standardized ileal digestiblility
[g]ME = Metabolizable energy
[h]Kcal = Kilocalories (1,000 cal)

Results and Discussion

The growth performance responses of growing-finishing pigs to varying dietary concentrations of DDGS, RAC inclusion and dietary DDGS withdrawal are provided in Table 3. During the Grower 1 feeding period (day 0 to 14), increasing dietary DDGS concentration resulted in a linear decrease in ADG ($P = 0.002$); however, DDGS concentration did not affect ADFI, G:F, BF and LMA ($P = 0.613$, 0.128, 0.408, and 0.855, respectively).

For the Grower 2 period (day 14 to 35), there was no difference among treatments for ADG, ADFI, and G:F ($P > 0.05$). In general, pigs fed 40% DDGS during Grower 1 exhibited decreased ADG compared to pigs fed the control diet; however, during Grower 2 that pattern was reversed. No differences in BF or LMA were recorded among treatments at the end of Grower 2 (day 34; $P = 0.674$ and 0.565 respectively).

For the Finisher 1 feeding period (day 35 to 56), treatments did not affect ADG, ADFI, or G:F ($P = 0.745$, 0.713, and 0.290, respectively). The inclusion of dietary DDGS did not affect LMA at the end of the Finisher 1 phase ($P = 0.349$). Unlike previous phases, there was a linear reduction in BF in response to dietary DDGS inclusion ($P = 0.048$). The lowest BF was recorded for 40% DDGS (0.61 in) and the greatest was recorded by 0% DDGS (0.79 in).

During Finisher 2 feeding phase (day 56 to 98), there was no effect of DDGS inclusion on ADG. Average daily gain, ADFI, and G:F were not affected by DDGS withdrawal during the last feeding period ($P = 0.187$, 0.274, and 0.312, respectively). At day 98, BF and LMA were not affected by treatment ($P = 0.804$ and 0.586 respectively). Final body weight was not affected by dietary treatment (DDGS, RAC or DDGS withdrawal; $P = 0.75$).

Overall, for ADG, ADFI and G:F there were no effects of dietary DDGS concentration observed ($P > 0.05$). Numeric trends show a slight increase in ADG in response to RAC. The greatest ADG overall (2.29 lb) was observed for the 15% dietary DDGS supplemented with 4.5 ppm of RAC for four weeks prior harvesting; furthermore, pigs receiving this dietary treatment also exhibited the greatest G:F (0.36 lb/lb).

Our results are consistent with previous findings reported in the literature that reported no changes in growth performance with up to 15% dietary inclusion of DDGS. We showed that it is possible to feed up to 40% dietary DDGS inclusion throughout the growing-finishing period and

(Continued on next page)

Table 3. Response and effect of distillers dried grains with solubles (DDGS) inclusion and ractopamine (RAC) on average daily gain (ADG), average daily feed intake (ADFI), gain to feed ratio (G:F), body weight (BW), and longissimus muscle area (LMA) of growing-finishing pigs.

Treatment	1	5	2	6	3	7	4	8						
DDGS, % for G1, G2, and F1[a]	0	0	15	15	40	40	40	40						
DDGS, % for F2[b]	0	0	15	15	40	40	0	0						
RAC, ppm	0	4.5	0	4.5	0	4.5	0	4.5						
										P-value				
Item									SEM[c]	TRT[d]	L[e]	Q[f]	RAC	W[g]
No. of pigs	5	5	5	5	5	5	5	5						
Grower 1 (day 0 to 14)														
BW (day 0), lb	67.61	65.22	70.12	64.65	67.52	64.65	66.28	66.90	1.629	0.378	0.977	0.535		
LMA (day 0), in^2	1.68	1.64	1.77	1.66	1.72	1.71	1.86	1.67	0.092	0.744	0.341	0.737		
BF (day 0), in	0.36	0.40	0.39	0.33	0.38	0.35	0.37	0.36	0.028	0.618	0.333	0.692		
ADG, lb	2.09	2.01	2.07	2.18	1.74	1.76	1.96	1.92	0.082	0.007	0.002	0.053		
ADFI, lb	4.17	4.01	4.32	4.08	4.39	3.64	4.23	4.06	0.260	0.613	0.878	0.622		
G:F, lb/lb	0.50	0.50	0.48	0.53	0.42	0.48	0.47	0.47	0.024	0.128	0.040	0.416		
BW (day 14), lb	97.20	93.49	99.23	95.21	92.04	90.67	100.37	93.89	2.115	0.297	0.075	0.116		
BF (day 14), in	0.42	0.44	0.41	0.35	0.33	0.40	0.39	0.35	0.035	0.408	0.046	0.427		
LMA (day 14), in^2	2.23	2.15	2.29	2.21	2.19	2.12	2.24	2.13	0.111	0.855	0.746	0.434		
Grower 2 (day 14 to 35)														
ADG, lb	2.27	2.21	2.15	2.36	2.18	2.32	2.32	2.40	0.139	0.465	0.504	0.916		
ADFI, lb	5.71	5.67	5.60	5.78	5.38	5.58	5.76	6.24	0.240	0.672	0.817	0.956		
G:F, lb/lb	0.40	0.39	0.38	0.41	0.40	0.42	0.40	0.39	0.015	0.497	0.370	0.940		
BW (day 35), lb	144.87	139.89	144.34	144.82	144.52	138.58	142.53	144.74	3.766	0.512	0.598	0.401		
BF (day 35), in	0.56	0.55	0.59	0.53	0.49	0.57	0.54	0.57	0.048	0.674	0.705	0.838		
LMA (day 35), in^2	3.55	3.52	4.07	3.65	3.34	3.46	3.79	3.49	0.223	0.565	0.833	0.060		
Finisher 1 (day 35 to 56)														
ADG, lb	2.03	2.18	2.32	2.36	2.09	2.29	2.18	1.90	0.192	0.745	0.859	0.154		
ADFI, lb	6.15	6.50	6.57	7.01	6.37	7.01	6.77	6.79	0.404	0.713	0.524	0.274		
G:F, lb/lb	0.33	0.33	0.35	0.34	0.33	0.36	0.32	0.27	0.015	0.290	0.305	0.336		
BW (day 56), lb	187.69	185.75	193.07	194.70	182.09	187.73	188.53	184.60	6.593	0.750	0.672	0.163		
BF (day 56), in	0.74	0.79	0.66	0.72	0.61	0.71	0.62	0.69	0.062	0.680	0.048	0.510		
LMA (day 56), in^2	4.82	4.76	5.08	5.23	4.34	4.79	5.41	4.43	0.314	0.349	0.699	0.215		
Finisher 2 (day 56 to 98)														
ADG, lb	1.85	1.89	2.12	2.26	1.98	1.87	2.00	2.25	0.146	0.702	0.336	0.065	0.436	0.187
ADFI, lb	6.24	6.46	6.73	6.97	6.64	6.57	6.59	7.36	0.326	0.411	0.248	0.161	0.217	0.274
G:F, lb/lb	0.29	0.29	0.31	0.32	0.30	0.28	0.30	0.30	0.013	0.453	0.176	0.017	0.880	0.312
BW (day 98), lb	265.39	265.31	282.02	289.83	265.44	266.19	272.49	279.20	11.365	0.734	0.822	0.035	0.640	0.384
BF (day 98), in	0.92	0.93	0.96	0.87	0.98	0.82	0.86	0.87	0.082	0.905	0.804	0.956	0.319	0.670
LMA (day 98), in^2	6.45	6.35	6.80	6.82	5.62	6.50	6.79	6.48	0.499	0.468	0.586	0.235	0.728	0.257
Overall (day 0 to 98)														
ADG, lb	2.07	2.07	2.16	2.29	2.01	2.03	2.12	2.12	0.106	0.678	0.159	0.174		
ADFI, lb	5.58	5.67	5.80	5.98	5.56	5.91	5.84	6.11	0.187	0.345	0.386	0.192		
G:F, lb/lb	0.34	0.34	0.35	20.36	0.33	0.34	0.34	0.33	0.011	0.637	0.134	0.162		
Carcass characteristics														
Hot carcass weight, lb	202.28	216.60	197.94	207.32	200.84	225.90	201.24	212.24	10.75	60.523	0.980	0.033	0.564	0.297
DP[h], %	74.15	73.51	74.66	73.74	72.88	73.94	74.08	73.81	0.006	0.485	0.608	0.320	0.610	0.312

[a]G1 = Grower 1; G2 = Grower 2; F1 = Finisher 1
[b]F2 = Finisher 2
[c]SEM = Standard error of the mean
[d]TRT = Treatment
[e]L = Linear
[f]Q = Quadratic
[g]W = Withdrawal
[h]DP = Dressing percentage. DP = (live weight/hot carcass weight) × 100

maintain growth performance. Unexpectedly, we did not detect an effect of RAC on growth performance. The data from this experiment do not support the concept that the withdrawal of DDGS at the end of the growing-finishing period results in improved growth performance. Additional work is underway to determine the effects of DDGS supplementation, withdrawal, and RAC on carcass and meat quality.

Conclusions

Results of this experiment suggest that growth performance of barrows from the University of Nebraska–Lincoln herd was maintained as dietary DDGS inclusion increased from 0 to 40%. The withdrawal of DDGS during the last feeding phase or RAC supplementation did not result in altered growth performance.

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Economy of Adding Fibrous Feedstuffs to Sow Gestation Diets

Producers may be able to improve the profitability of their operation by using fibrous feed ingredients in sow gestation diets.

Duane E. Reese
Allen Prosch[1]

Summary

A previous summary of research results indicated that sows fed high-fiber diets during gestation weaned 0.3 more pigs/litter on the average than sows fed lower-fiber, grain-based diets. Gestation diets containing 18% soybean hulls, 46% distillers dried grains with solubles (DDGS), 34% wheat midds, 25% wheat bran, 23% alfalfa meal, 25% sugar beet pulp, or 45% oats provide sows about 350 g/day of neutral detergent fiber (NDF), which may be sufficient to increase litter size weaned by 0.3 pigs per litter. An economic analysis suggests that feeding a diet containing these sources of NDF would increase sow feed ingredient costs from 0 to $22.35 per sow per gestation period compared to feeding a corn-soybean meal diet. No improvement in litter size at weaning was required to justify feeding DDGS at the ingredient prices assumed in this analysis. Small improvements in litter size (0.16 to 0.24 pigs per litter) would be necessary to justify feeding soybean hulls or wheat midds during gestation. Producers may be able to improve the profitability of their operation by using fibrous feed ingredients in sow gestation diets.

Introduction

In the pork industry, high-fiber, low energy-dense diets are best suited for gestating sows. Gestating sows utilize fiber better than growing pigs, and they have a high feed intake capacity relative to their energy requirement during gestation. Results from a review of 24 research studies on the effects of providing high-fiber diets to sows during gestation was published in the *2008 Nebraska Swine Report*. That review suggested sows fed high-fiber diets during gestation weaned 0.3 more pigs per litter than those fed low-fiber diets.

Table 1. Diets for gestation sows (as-fed basis).

	Diet							
Ingredient, lb	Corn-soy	18% soybean hulls	46% DDGS[a]	34% Wheat midds	25% Wheat bran	23% Alfalfa meal	25% Beet Pulp	45% Oats
Corn	1662	1367	858	1093	1263	1313	1214	855
Soybean meal, 46.5% CP	254	197	150	152	169	166	212	178
Soybean hulls		360						
DDGS			917					
Wheat midds				679				
Wheat bran					490			
Alfalfa meal						462		
Beet pulp							502	
Oats								890
Dicalcium phosphate, 18.5% P	47	43	24	33	35	36	45	41
Limestone	14	10	28	20	20		4	13
Salt	10	10	10	10	10	10	10	10
Vitamin/trace mineral mix	13	13	13	13	13	13	13	13
Daily Intake								
Feed, lb	4.1	4.3	4.1	4.3	4.5	4.6	4.4	4.5
Metabolizable energy, Mcal[b]	6.1	6.1	6.1	6.1	6.1	6.1	6.1	6.1
Lysine (total), g	11.0	11.8	12.8	10.8	11.6	12.3	12.0	11.3
Lysine (SID), g	9.3	9.3	9.3	9.3	9.3	9.3	9.3	9.3
Calcium, g	15.8	15.8	15.9	15.9	17.2	16.7	15.9	15.8
Phosphorus (total), g	14.0	13.4	13.1	15.9	17.2	13.2	13.5	14.3
Phosphorus (available), g	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0
Neutral detergent fiber, g	173	350	350	350	350	350	350	350
Calculated Analysis, %								
Lysine, total	0.59	0.60	0.69	0.55	0.57	0.59	0.60	0.55
Lysine, SID	0.50	0.47	0.50	0.48	0.46	0.44	0.47	0.45
Calcium, %	0.85	0.80	0.86	0.82	0.85	0.80	0.80	0.77
Phosphorus, total	0.75	0.68	0.71	0.82	0.85	0.63	0.68	0.69
Phosphorus, available	0.48	0.46	0.49	0.47	0.44	0.43	0.45	0.44

[a]Distillers dried grains with solubles
[b]Metabolizable energy (Mcal/lb) and NDF (%) values for corn, soybean meal, soybean hulls, DDGS, wheat midds, wheat bran, alfalfa meal, beet pulp, and oats were 1.56, 1.52, 1.06, 1.55, 1.38, 1.03, 0.75, 1.13, 1.23, and 9.6, 10.2, 56.4, 30.5, 35.6, 42.1, 41.2, 42.4, 27, respectively

High corn and soybean meal prices have pork producers searching for alternative feed ingredients. Fibrous feed ingredients in sow gestation diets should be part of that search. When making a decision to add fibrous feed ingredients to gestation diets, it is important to conduct an economic analysis. The economic analysis presented in this paper included consideration for feed ingredient costs and weaned pig value; costs associated with ingredient storage, feed handling, and manure disposal were not included.

Procedures

Eight corn/soybean meal-based gestation diets were formulated (Table 1). One diet (corn-soy) contained no additional fiber; the remaining seven diets contained additional fiber through the addition of either 18% soybean hulls, 46% DDGS, 34% wheat midds, 25% wheat bran, 23% alfalfa meal, 25% sugar beet pulp, or 45% oats. All diets were formulated to provide sows with similar daily amounts of metabolizable energy, standardized ileal digestible (SID) lysine, calcium, and available phosphorus by altering ingredient composition and daily feed intake. Each of the high-fiber diets was formulated to provide 350 g/day of neutral detergent fiber (NDF), an amount previously suggested that may be necessary to elicit a positive litter size response *(1997*

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Nebraska Swine Report). Total sow feed ingredient cost for a 110-day feeding period was estimated for each diet.

Results and Discussion

Variation in the cost of the complete diets and in the total sow feed ingredient expense among the eight feeding programs was observed (Table 2). The total feed expense per sow per 110-day period for the diets with various sources of additional fiber increased with a range of $0.00 to $22.07/sow compared to the corn-soybean meal diet. The cost of feeding the 46% DDGS diet was similar to that for the corn-soybean meal diet. Expense incurred from feeding the 34% wheat midds or 18% soybean hulls diet was $3.29 and $4.76 per sow more than that for the corn-soybean meal diet. The 25% wheat bran, 23% alfalfa meal, and 25% beet pulp and 45% oats feeding programs were considerably more expensive than the corn-soybean meal diet program.

When lower energy, fibrous feedstuffs are added to the diet, sows often must be provided more feed to meet their daily metabolizable energy requirement. Feeding a gestation diet containing 34% wheat midds or 18% soybean hulls resulted in 4.6% greater feed usage compared to feeding the corn-soybean meal diet (Table 2). Feeding a diet containing 25% wheat bran, 23% alfalfa meal, 25% beet pulp, or 45% oats increased feed usage by 9, 12, 7, and 10%, respectively compared to feeding the corn-soybean diet. The amount of feed for sows fed the 46% DDGS diet was similar to that for those fed the corn-soybean meal diet. Therefore, it is important to compare total feed ingredient cost per sow per period of time rather than ingredient cost per ton of feed when evaluating the economics of feeding many high-fiber diets to gestating sows.

In the event a producer faces additional sow feed expense, such as revealed in the feeding of all fibrous ingredients except DDGS in this analysis, additional value must be generated in order to justify the extra feed expense. Litter size improvement can represent increased value. The change in litter size at weaning needed to offset additional sow feed ingredient expense is presented in Table 3. The calculations are based on pig values at weaning of $20, 25, 30, 35, 40, and 45 per pig. With DDGS being the exception, the use of all fibrous feedstuffs required an increase in litter size ranging from 0.11 to 1.12 pigs per litter to pay for the extra sow feed expense incurred. Soybean hulls and wheat midds required the least litter size improvement (0.16 to 0.24 depending on pig value); wheat bran required the greatest litter size improvement to offset the additional feed expense associated with feeding the various fiber sources.

Based on the results of the original review presented in the *2008 Nebraska Swine Report*, it is somewhat reasonable to expect a litter size improvement at weaning. A total of 34 comparisons evaluating litter size at weaning were made between sows fed control and high-fiber diets; in 19 (56%) of those comparisons an increase in litter size was observed while in 12 (35%) a decrease was observed. On average, 0.3 more pigs were weaned per litter. A larger improvement in litter size at weaning (0.6 pigs per litter) was observed in studies where sows were fed high-fiber diets over multiple reproductive cycles. This implies that, to ensure an improvement in litter size from feeding fiber, fiber-feeding must be initiated before mating.

The results in Table 3 are valid for the ingredient prices used in this analysis only. Given the price volatility the feed ingredient market has experienced recently, producers are advised to frequently evaluate prices for high-fiber feed ingredients for possible inclusion in sow gestation diets. The diets in Table 2 could serve as the basis for evaluating the economic feasibility of feeding fibrous ingredients to gestation sows.

The amount of manure solids produced from feeding these high-fiber diets would probably increase in proportion to the extra amount of feed provided, which could be a problem in some manure disposal systems. Some producers report that the undigested portion of the hull from oats is particularly a nuisance to remove from manure storage devices.

Table 2. Feed ingredient costs and feed usage estimates for various gestation feeding programs.

Item	Diet							
	Corn-soy	Soybean hulls	DDGS[a]	Wheat midds	Wheat bran	Alfalfa meal	Beet pulp	Oats
Feed cost/ton, $[b]	190	200	189	197	265	213	226	238
Feed cost/sow/period, $[b,c]	42.94	47.70	42.39	46.23	65.29	54.05	54.62	59.14
Difference in sow feed expense (vs. corn-soy), $/sow/period		4.76	0	3.29	22.07	11.10	11.71	16.20
Gestation feed usage, lb/sow/period[c]	452	478	449	468	492	508	483	498

[a]Distillers dried grains with solubles.
[b]Ingredient prices used were corn $3.80/bu; soybean meal $290/ton; soybean hulls $225/ton; DDGS $170/ton; wheat midds $197/ton; wheat bran $490/ton; alfalfa meal $290/ton; beet pulp $300/ton; oats $262/ton; dicalcium phosphate $1,050/ton.
[c]Period = 110 days; Daily metabolizable energy intake = 6.1 Mcal.

Table 3. Change in number of pigs weaned/litter needed to offset extra sow feed ingredient expense per 110-day gestation period.

Diet	Value of a pig at weaning, $/pig					
	20	25	30	35	40	45
Corn-soy						
Soybean hulls	0.24	0.19	0.16	0.14	0.12	0.11
DDGS[a]	-0.03	-0.02	-0.02	-0.02	-0.01	-0.01
Wheat midds	0.16	0.13	0.11	0.09	0.08	0.07
Wheat bran	1.12	0.89	0.74	0.64	0.56	0.50
Alfalfa meal	0.56	0.44	0.37	0.32	0.28	0.25
Beet pulp	0.58	0.47	0.39	0.33	0.29	0.26
Oats	0.81	0.65	0.54	0.46	0.40	0.36

[a]Distillers dried grains with solubles.

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Nutrition During Gilt Development and Genetic Line Affect Reproductive Rate Through Parity 1

Twenty-five percent energy restriction during development delays sexual development of gilts but has no effect on reproductive rate of those reaching sexual maturity.

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Summary

Effects of allowing gilts ad libitum access to feed until breeding age or developing them with 25% energy restriction from 123 days of age to breeding on reproductive success through parity 1 were studied with a total of 639 gilts of two lines that differ in lean growth and reproduction. Gilts of the two lines had common sires, an industry maternal line, but dams were from different populations. One line of gilts, LW x LR, represented standard industry Large White x Landrace cross females. The other gilts, L45X, were daughters of Nebraska selection Line 45 that has been selected 27 generations for increased litter size with additional selection for increased growth and decreased fat in the last seven generations. More L45X than LW x LR gilts (95 vs. 88%, P < 0.01) and more gilts developed with ad libitum intake than with restricted intake (96 vs. 86%, P < 0.01) expressed puberty by 226 days of age. For gilts that expressed puberty, mean age at puberty was 6 days less (P < 0.01) for L45X than LW x LR gilts, but did not differ between gilts on the two developmental regimens. For all gilts, the likelihood of expressing puberty increased with increasing weight at 123 days of age. It was also greater for gilts that attained heavier weights with greater backfat at 226 days of age, but the effect varied among lines and gilt developmental regimens. Increasing weight and backfat at 226 days of age increased the likelihood of producing a parity 1 litter for L45X gilts developed with restricted feeding, but not for other groups. Number of live born pigs per litter was affected by line, being greater for L45X gilts (P < 0.05), but not by gilt developmental regimen. Neither line nor gilt developmental regimen affected maternal ability as measured by number and weight of pigs weaned. A 25% energy restriction during gilt development decreases the likelihood that gilts express estrus by 226 days of age, but has little effect on subsequent reproductive performance.

Introduction

It has been shown in several species that prolonged periods of energy restriction initiated postweaning, without limiting other nutrients, often results in increased longevity that is approximately proportional to the level of restriction. However, reallocation of nutrients often occurs such that animals cannot combine high rates of fecundity with extended lifespans. Research with mice has shown that this outcome is not always true. A recent publication of one experiment contains data showing that female mice restricted in energy intake postweaning lived longer without a reduction in reproductive rate.

Today's commercial gilts are often managed to achieve weights of at least 136 kg (300 lb) with adequate backfat at breeding, although the amount of backfat that is adequate is not well defined. These targets are often achieved with management practices that include ad libitum access to feed. However, consistent with the findings in mice, a series of reports containing data from experiments at the USDA Meat Animal Research Center demonstrated that moderate feed restriction during prepubertal development of gilts may increase reproductive efficiency through first parity.

Optimum gilt development regimens may depend on the prolificacy and lean growth rate of the genetic line. We initiated an experiment with the overall objective of estimating the effects of 25% restriction of energy vs. ad libitum access to feed from 123 days of age to breeding on reproduction and longevity through parity 4 of females of two lines that differ in rate of lean growth and litter size. The experiment was done in four replications with a total of 661 gilts. The *2008 Nebraska Swine Report* contained articles summarizing effects of line and gilt developmental regimen on growth of gilts to 226 days of age and subsequent reproductive performance of females through parity 4 for gilts of replications 1 to 3. Since that report, replication 4 gilts produced parity 1 litters, providing a complete dataset through parity 1. The objective of this experiment is to summarize effects of line and energy restriction on reproductive performance through parity 1. Effects of variation in measures of growth (weight, backfat, and longissimus muscle area at different ages) on the likelihood of expressing puberty and producing a parity 1 litter are also presented.

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Materials and Methods

Gilt populations

Two populations of gilts were used. One was the Large White x Landrace crossbred female used regularly in the University of Nebraska–Lincoln swine nutrition program. The project gilts were the progeny of Large White-Landrace cross sows that had been inseminated with semen of industry maternal line (L_M) boars and are designated as LW x LR cross. Gilts that were progeny of UNL selection Line 45 sows that had been inseminated with semen of the same L_M boars used to produce LW x LR gilts comprised the other population. These gilts are designated as L45X. Line 45 has been selected 27 generations for increased litter size with additional selection for increased growth and decreased backfat in the last seven generations. Based on previous data, L45X gilts were expected to be more prolific than LW x LR gilts but to also have somewhat slower growth and greater backfat thickness.

Gilt Management and Dietary Regimens

The experiment was done in four replications in which project gilts were born in batches during December 2004 and January 2005 (Rep 1), May 2005 (Rep 2) and November 2005 (Rep 3), and May and June 2007 (Rep 4). A total of 661 gilts began the experiment (157 to 185 gilts per replication) at 60 days of age; 639 of them completed the growth phase of the experiment that ended at 226 days of age.

Dams of project gilts were managed alike during the farrowing/lactation period. After weaning, all gilts were managed alike in the nursery until approximately 60 days of age (21 kg (46 lb)). They were then moved to the grow-finish facility where they were penned (10/pen) by line-treatment designation. All gilts were allowed ad libitum access to a corn-soybean meal-based diet and were managed alike until 123 days of age. A 3-phase growing-finishing diet was used: phase 1, 1.15% lysine (60 days to 80 lb); phase 2, 1.0% lysine (80 to 130 lb); and phase 3, 0.90% lysine (130 lb to 123 days).

At 123 days of age, pens of gilts on the ad libitum regimen (A) were allowed ad libitum access to a corn-soybean meal based diet (0.70% lysine, 0.70% Ca, 0.60% P) until they were moved into the breeding barn. Gilts treated with the restricted intake regimen (R) received a corn-soybean meal based diet at approximately 75% of the energy intake as A-fed gilts until moved into the breeding barn. Energy restriction was achieved by predicting intake with a quadratic equation of average daily feed intake on body weight of A-fed gilts. The predicted ad libitum intake (based on the projected body weight for the upcoming two-week period) was multiplied by 0.75 to determine the daily feed intake for R gilts. The diet contained 0.93% lysine, 1.0% Ca, and 0.80% P. All vitamins and minerals, except selenium, were increased so that daily intake of these nutrients per unit of body weight was expected to be equal for gilts on both diets. Additional details of the diets and management are in two articles in the *2007 Nebraska Swine Report*.

During the growing period, gilts were weighed and backfat and longissimus muscle area were recorded every 14 days until final measurements were recorded at an average age of 226 days. Beginning at approximately 140 days of age, gilts were moved by pen to an adjacent building where boar exposure and estrus detection occurred. Date of first observed estrus and each additional estrus were recorded.

Breeding and Lactation Management

Gilts in good health and that could be mated at third or later estrus during a predetermined breeding period were identified as breeders and moved to the breeding barn at approximately 230 days of age. Breeding commenced approximately 10 days later. A breeding period of 25 days (Rep 1), 24 days (Rep 2), 26 days (Rep 3), and 28 days (Rep 4) was used to match the unit's production schedule. Gilts were checked twice daily for estrus and were inseminated each day that they were observed in estrus. Insemination was with semen of boars from a commercial terminal sire line. Gilts were in pens of approximately eight per pen until inseminated and then were moved to gestation stalls. Gilts that did not express estrus, those that were mated but diagnosed open with an ultrasound pregnancy test 50 days postbreeding, and those that were diagnosed pregnant but did not farrow a litter were culled. Lame gilts and those in poor health also were culled.

While in the breeding barn and during gestation, all gilts were fed a standard corn-soybean meal based diet (13.8% protein, 0.66% lysine) at the rate of 4.0 lb daily until 90 days of gestation when feed intake was increased to 5.0 lb daily. At approximately 110 days of gestation, females were placed in farrowing crates in rooms of 12 crates per room and fed 6 lb daily of a corn-soybean meal based lactation diet (18.5% protein, 1.0% lysine). Sows were provided only a small amount of feed on the day they farrowed, 6 lb during the second day and 10 lb during the third day of lactation, and then were given ad libitum access to feed. Total number and number of live pigs were recorded for each sow. Pigs were fostered among litters without regard to line or gilt developmental regimen to reduce variation in number nursed per sow. Litters were weaned at an average age of approximately 17 days and number weaned and total litter weight were recorded.

Traits and Data Analysis

Gilts completing the growth test were coded as 0 if they had not expressed a pubertal estrus and 1 if they had. Then, based on females designated for breeding, they were coded as 1 if they farrowed a litter at parity 1and 0 if not. These scores, which are measures of success/failure to reproduce, were fitted with general linear models designed for binomial data to determine the importance of line, gilt treatment, and interaction of line with treatment. Then, weight, backfat, and longissimus muscle area at 123

Table 1. Number of gilts that did and did not express pubertal estrus, number designated as breeders, and number of breeders that did and did not produce a parity 1 litter.

		Expressed pubertal estrus		Parity 1 litter		
Line	Trt[1]	Yes	No	Breeders	Yes	No
LW x LR	A	159	15	139	107	32
LW x LR	R	133	38	123	97	26
L45X	A	143	4	129	112	17
L45X	R	132	15	119	96	23
Total		567	72	510	412	98

[1]A = ad libitum regimen, R = restricted intake regimen

Table 2. Mean reproductive rates for Large White x Landrace (LWxLR) and Line 45 cross (L45X) gilts developed with ad libitum feeding (A) or 25% energy restriction (R).

		Pr[1] Pubertal estrus[1]		Age puberty[2]		Pr P1 litter		Total born/litter[3]		Live born/litter[4]	
Line	Trt	Avg	SEM	Avg	SEM	Avg	SEM	Avg	SEM	Avg	SEM
LwxLR	A	0.93	0.05	177.8	3.7	0.73	0.04	12.65	0.36	11.04	0.34
LwxLR	R	0.80	0.05	176.2	3.7	0.75	0.04	12.31	0.38	11.00	0.35
L45X	A	0.98	0.01	170.0	3.7	0.84	0.04	12.56	0.36	11.37	0.33
L45X	R	0.90	0.03	172.1	3.7	0.80	0.04	13.54	0.38	12.21	0.35
LwxL	R	0.88	0.03	177.0	3.5	0.74	0.03	12.48	0.29	11.02	0.25
L45	X	0.95	0.02	171.0	3.5	0.82	0.03	13.05	0.29	11.79	0.25
	A	0.96	0.01	173.9	3.5	0.79	0.03	12.61	0.27	11.20	0.24
	R	0.86	0.03	174.2	3.5	0.77	0.03	12.92	0.29	11.61	0.25

[1]Pr = probability
[2]Line, P = 0.006; trt, P = 0.0001
[3]Line x trt, P = 0.05
[4]Line, P = 0.03

Table 3. Mean number weaned and litter weaning weight for Large White x Landrace (LWxLR) and Line 45 cross (L45X) gilts developed with ad libitum feeding (A) or 25% energy restriction (R).

		Number weaned per litter[1]		Litter weaning weight, kg[2]	
Line	Trt	Mean	SEM	Mean	SEM
LwxLR	A	9.38	0.22	45.36	1.20
LwxLR	R	9.04	0.22	43.18	1.23
L45X	A	9.18	0.21	42.91	1.19
L45X	R	9.24	0.22	45.62	1.24
LwxLR		9.46	0.28	45.45	1.70
L45X		9.30	0.28	45.26	1.70
	AL	8.90	0.28	40.37	1.67
	R	9.19	0.30	45.99	1.81

[1]Line, P = 0.23; trt, P = 0.81
[2]Line, P = 0.21, Trt, P = 0.12

and at 226 days of age were fitted as co-variables to determine how they affected the likelihood of expressing puberty or farrowing a litter. Effects of age at puberty on likelihood of farrowing a litter also were estimated. Covariate effects were estimated by predicting mean probabilities at various levels of the covariates and predicted means were graphed to illustrate relationships.

Results

A total of 639 gilts completed the growth phase of the experiment. Of these gilts, 567 expressed an estrus by 226 days of age and 510 were designated as breeders. Of the 57 gilts that expressed estrus but were not designated as breeders, 20 were randomly culled, five within each line x treatment combination, to reduce breeding numbers to fit the production capacity. The remaining 37 gilts were culled for health or because they expressed estrus so late that they could not be mated at third or later estrus. This culling was not related to line or treatment. Distributions of gilts with a pubertal estrus and that farrowed a litter across lines and treatments are in Table 1.

Table 2 contains the probability that gilts expressed estrus, mean age at puberty for those that did express estrus, the probability that gilts designated for breeding produced a parity 1 litter, and mean litter size for those that farrowed. Both line and gilt developmental regimen significantly affected the proportion of gilts that expressed a pubertal estrus. The probability that gilts expressed a pubertal estrus was 0.95 for Line 45X gilts and 0.88 for LW x LR gilts ($P = 0.006$). The probability of expressing pubertal estrus also was greater for gilts developed with ad libitum access to feed than those developed with restricted energy intake (0.96 vs. 0.86, $P = 0.0001$). The interaction was not significant as effects of gilt developmental regimen were similar for both lines.

For those gilts designated as breeders, the probability of producing a litter was greater for L45X than for LW x LR gilts, although the difference was not significant ($P = 0.33$). For gilts designated as breeders, the developmental regimen they had been on did not affect the likelihood they farrowed a parity 1 litter.

Interaction of line and gilt developmental regime existed for total number of pigs farrowed per litter, but not for live pigs per litter. Total born per litter was greater for LW x LR gilts developed with ad libitum access to feed than when developed with restricted intake, but the reverse occurred for L45X gilts as those developed with restricted intake farrowed more total pigs. This interaction did not exist for live pigs per litter, but L45X gilts produced more live pigs than LW x LR gilts (11.79 vs. 11.02, $P = 0.03$). Gilt developmental regimen did not affect live pigs per litter.

Number of pigs weaned per litter and litter 17-day weaning weight were

(Continued on next page)

standardized for the number of pigs after pigs were fostered among litters, and thus do not reflect line and treatment differences in live pigs per litter (Table 3). After this standardization, neither line nor gilt development regimen significantly affected number or weight of pigs at weaning, even though litter weaning weight was 14% greater ($P = 0.12$) for gilts developed with restricted energy intake than for those developed with ad libitum intake. Thus, given an opportunity to raise the same number of pigs, gilts of the two lines developed with either regimen did not differ greatly in maternal ability.

Relationships between weight at 123 days of age and the probability of expressing a pubertal estrus are illustrated in Figure 1. Response was curvilinear, but in general, the probability of expressing pubertal estrus increased with increasing 123-day weight. The effect was greatest ($P = 0.04$) for L45X gilts developed with restricted energy intake. The effect was not significant in other groups ($0.10 \leq P \leq 0.15$), but trends were similar in that increasing weight was associated with greater probability of reaching puberty. Therefore, heavier gilts at 123 days of age had a greater chance of attaining puberty by 226 days of age and when restricted in energy intake, heavier gilts at the start of the restriction period were more likely to attain puberty than lighter gilts, especially the L45X gilts. Backfat at 123 days was not associated with the likelihood of attaining puberty.

Final weights and scan backfats and longissimus muscle areas were recorded after estrus checking was terminated. However, weight and backfat at 226 days of age were related with the probability a gilt expressed estrus. A strong relationship with weight existed in each line by treatment class ($0.002 \leq P \leq 0.06$), but was greatest for L45X gilts developed with ad libitum access to feed. The probability of expressing estrus was less than 0.6 for L45X gilts that weighed less than 100 kg at 226 days of age. At all weights, the probability of having expressed puberty was 0.65 or greater for all other groups, but it increased with weight up to approximately 120 kg.

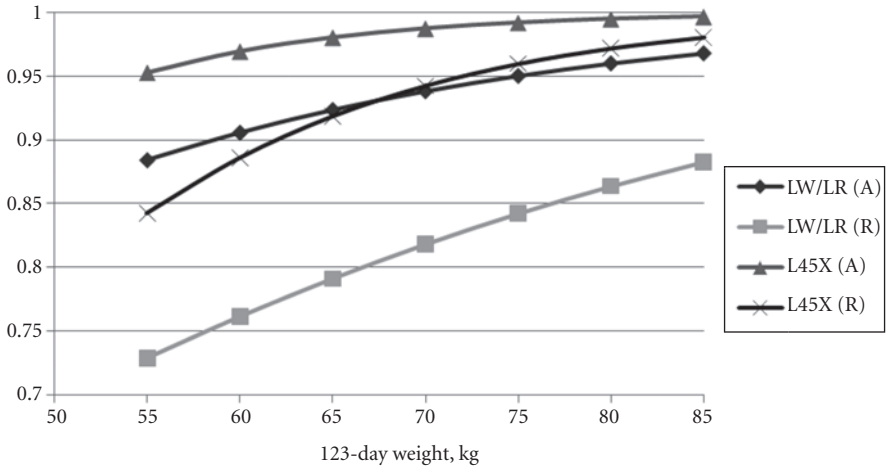

Figure 1. Effect of 123-day weight on probability of pubertal estrus.

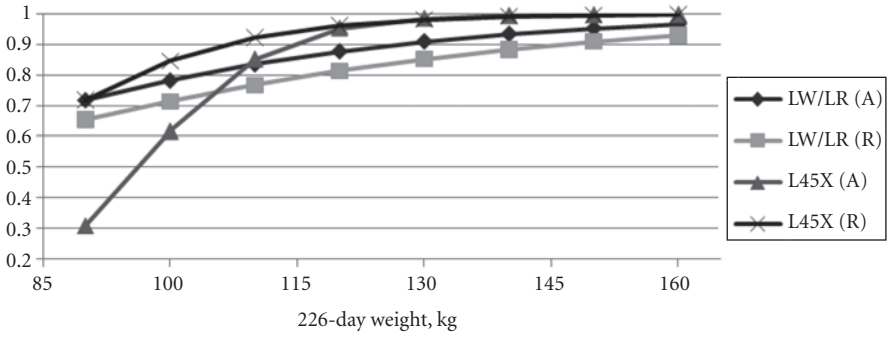

Figure 2. Effect of 226-day weight on probability of pubertal estrus.

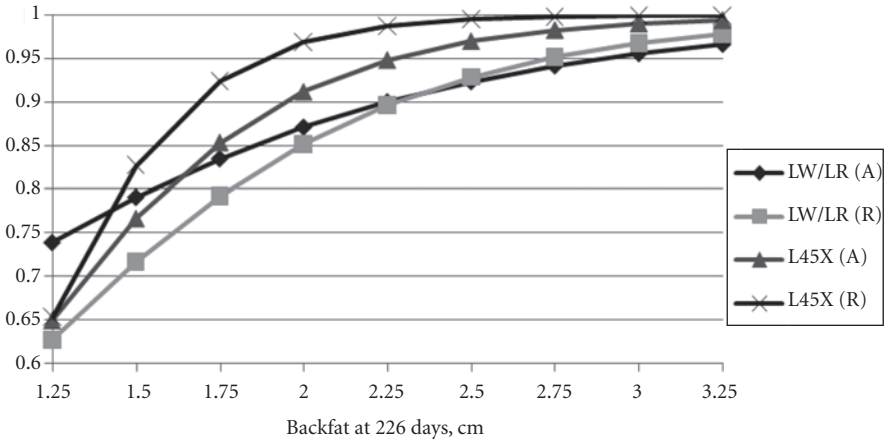

Figure 3. Effect of 226-day backfat on probability of pubertal estrus.

The relationship of backfat at 226 days with the probability of having expressed pubertal estrus was similar to that of 226-day weight (Figure 3) but was not significant for any class ($0.11 \leq P \leq 0.35$). The overall trend ($P = 0.11$) was that as backfat increased, the probability of having expressed estrus increased, but depth of backfat at the end of the growing

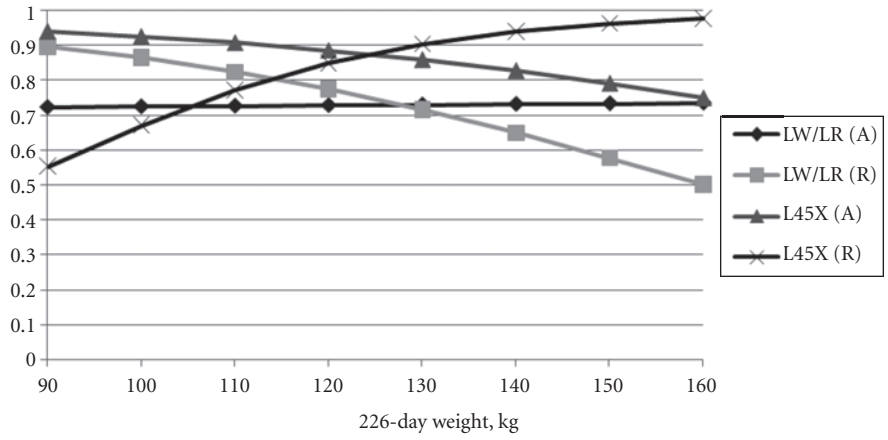

Figure 4. Effect of 226-day weight on the probability gilts produced a parity 1 litter.

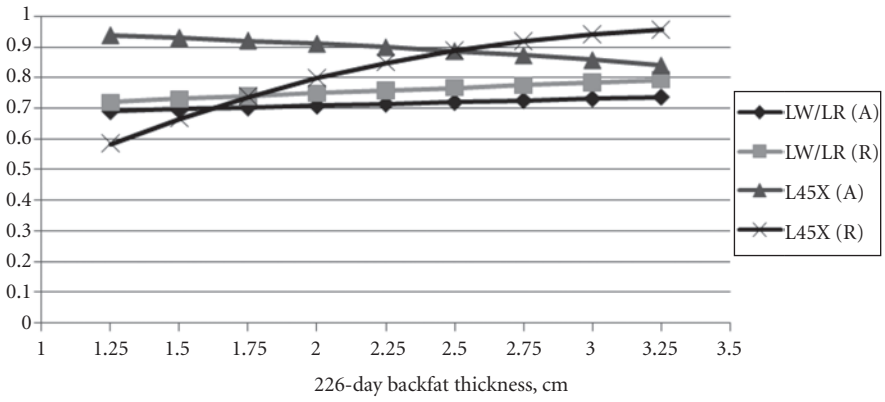

Figure 5. Effect of backfat at 226 days on probability of farrowing a litter.

period was not strongly associated with whether gilts attained puberty. Longissimus muscle area at 226 days was not related with whether gilts attained puberty in any group ($0.09 \leq P \leq 0.22$).

Relationships of 226-day weight and backfat with the probability a gilt farrowed a parity 1 litter are in Figures 4 and 5, respectively. The effect of weight was somewhat odd. For three groups, all except L45X gilts developed with restricted energy intake, the probability of farrowing a litter decreased with increasing weight. However, none of those relationships were significant ($.09 \leq P \leq 0.95$). For L45X gilts developed with energy restriction the probability of farrowing a litter increased ($P = 0.03$) with increasing 226-day weight. Similarly, increased backfat at 226 days was associated with increased probability of farrowing a litter only in L45X gilts developed with restricted energy intake ($P = 0.03$). The probability increased for this group with increasing backfat thickness. The effect was not significant in other groups ($P > 0.15$).

The implications of this research are that if gilts are targeted for breeding at second or later post-pubertal estrus and to farrow by 365 days of age, then the replacement gilt pool must be approximately 10% larger to produce a specified number of litters than if gilts are developed with ad libitum feed intake. Management of gilts early in life is also important. Increased 123-day weight, but not backfat or longissimus muscle area, was associated with increased likelihood of attaining puberty, regardless of which gilt development management regimen was used. For gilts that could be bred at second or later post-pubertal estrus, the likelihood they farrowed a litter and their litter size were not affected by the developmental regimen. It is commonly thought that the likelihood of reproductive success increases for gilts with greater weights and backfats when they enter the breeding herd. However, this result occurred only for L45X gilts developed with restricted energy intake. Therefore, weight and backfat of breeding age gilts are not good predictors of subsequent reproductive performance.

[1]Rodger K. Johnson and Phillip S. Miller are professors; Roman Moreno is a graduate student and research technician in the Animal Science Department; Matthew W. Anderson is manager; and Jeffrey M. Perkins, Kelsey Rhynalds, Trevor J. Glidden, Donald R. McClure, and Thomas E. McGargill are technicians at the UNL Swine Research Farm.

In Vivo and *In Vitro* Expression of Porcine Zinc Transporter (Znt) 1 mRNA

Zinc transporter (ZnT1) mRNA is differentially expressed *in vivo*, in weaned pig tissues, and *in vitro*, in response to additions of zinc sulphate or antibiotics.

Huyen Tran
Phillip S. Miller
Thomas E. Burkey[1]

Summary

Preliminary experiments were carried out to evaluate in vivo expression of zinc transporter (ZnT) 1 mRNA in a panel of tissues obtained from weaned pigs and to evaluate in vitro *changes in ZnT1 mRNA in a porcine jejunal epithelial cell line (IPEC-J2) treated with low (40 µM) or high (80 µM) concentrations of zinc sulphate ($ZnSO_4$) or, in a separate experiment, with low (50 µg/ml) or high (100 µg/mL) concentrations of antibiotics (as gentamicin; GENT). Lipopolysaccharide (LPS; 10 ng/mL) was included as a negative control in both experiments. Zinc transporter 1 mRNA was detected in all tissues evaluated with the greatest level of expression observed in the tonsil. A treatment × time interaction was not observed for IPEC-J2 cells treated with $ZnSO_4$; however, the addition of high $ZnSO_4$ tended ($P = 0.08$) to increase ZnT1 mRNA expression when means were averaged among all time points. Exposure of IPEC-J2 cells to GENT resulted in a significant treatment × time interaction ($P < 0.005$) with increases in ZnT1 mRNA observed at 3 (low GENT) and 6 (low and high GENT) hours post-treatment compared to CTL- or LPS-treated cells. This research indicates that ZnT1 mRNA is differentially expressed* in vivo*, in a panel of porcine tissues, and* in vitro*, in IPEC-J2 cells exposed to low or high concentrations of zinc or antibiotics.*

Introduction

Zinc (Zn) is a trace mineral which has a functional role in many metalloenzymes and is essential for normal growth because of the association with nucleic acids and protein synthesis. In pigs, a Zn-deficient diet has been reported to result in appetite reduction as a result of the lack of gustin, a protein involved in acuity. Conversely, pigs fed pharmacological levels of Zn experience improved growth performance and decreased incidence of diarrhea. In addition, other effects have been attributed to zinc, including anti-inflammatory and infectious resistance mediation, maintenance of membrane function and stability via stabilization of membrane structure or anti-oxidation, and protection of cells from free-oxygen radicals. Thus, it appears that Zn may have an effect on reducing pathogen colonization of cell surfaces, especially when fed at pharmacological concentrations.

Zinc transporter 1 (ZnT1) is a cellular zinc exporter which is predominantly located in intracellular compartments and plasma membrane to promote zinc efflux from the cells when cellular zinc is abundant. In addition, ZnT1 is distributed in a number of tissues but has been observed to be most abundantly expressed in the proximal small intestine of humans. Although the mechanism for ZnT1 expression in all species and tissues has not been fully elucidated, it has been shown that ZnT1 mRNA is regulated by zinc supplementation or deletion in human cultured cells or in pigs fed high concentrations of zinc. The induction of ZnT1 gene transcription has also been explained by the binding of the metal-specific transcription factor MTF-1 on two metal response elements in the ZnT1 promoter region in mice.

Most of the research evaluating the expression and regulation of ZnT1 has been conducted in humans and mice and little emphasis has been placed on studies evaluating ZnT1 expression and regulation in pigs. Previous work has demonstrated that expression of ZnT1 protein in the liver was greater in pigs fed 1,000 ppm Zn than pigs fed 2,000 ppm Zn (with or without phytase). It has also been observed that ZnT1 is involved in regulating Zn homeostasis in nursery pigs fed pharmacological concentrations of Zn. However, the mechanisms for ZnT1 mRNA expression in different types of tissues in weaned pigs or the *in vitro* response of ZnT1 mRNA in porcine gut epithelial cells to different culture conditions is not clear. Therefore, the objectives of the current experiments are to evaluate the *in vivo* expression of ZnT1 mRNA in a panel of tissues obtained from weaned pigs, and to assess the *in vitro* changes in ZnT1 mRNA in IPEC-J2 cells treated with low and high concentrations of $ZnSO_4$ or GENT.

Materials and Methods

In Vivo *Expression of ZnT1 mRNA in Porcine Tissues*

Four crossbred barrows (typical of commercial pigs), approximately 5 weeks of age, were used and the experimental protocol was approved by the University of Nebraska Institutional Animal Care and Use Committee of the University of Nebraska–Lincoln. Weaned pigs (n = 4) were sacrificed and tissues were collected at the UNL Veterinary Diagnostic Center. Tissues (spleen, jejunum, ileum, liver, tonsil, and thymus) were excised and immediately frozen in liquid nitrogen for subsequent analyses.

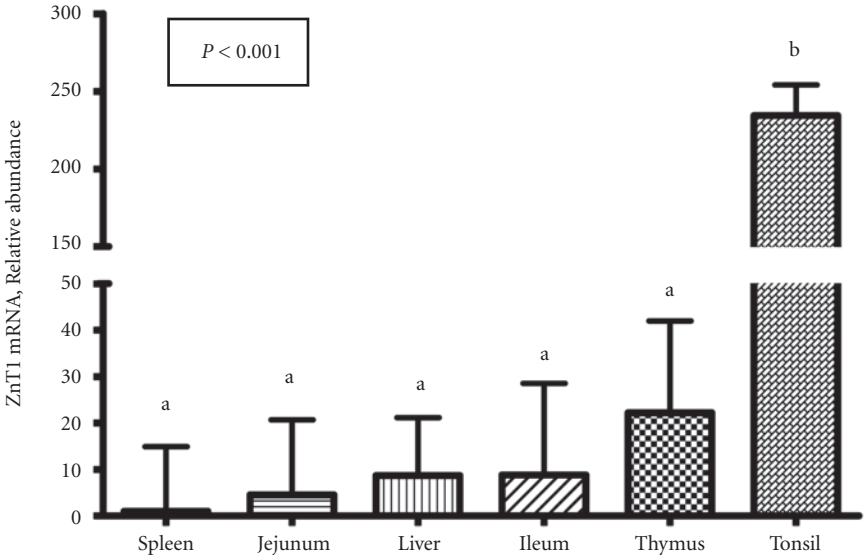

Figure 1. Relative abundance of zinc transporter (ZnT) 1 mRNA in porcine spleen, jejunum, ileum, liver, and thymus. Each bar represents the least squares mean (± SEM) of four observations. Bars without common superscripts differ ($P < 0.001$).

In Vitro *Expression of ZnT1 mRNA in IPEC-J2 Cells*

Two separate experiments were conducted using IPEC-J2 cells in order to characterize ZnT1 mRNA expression. The IPEC-J2 cells have been characterized previously and are nontransformed, jejunal epithelial cells derived from neonatal pigs and are maintained as a continuous culture. Cell cultures were maintained in DMEM-F12 growth medium supplemented with insulin/transferrin/Na selenite media supplement, epidermal growth factor, antibiotic and fetal bovine serum. For experimentation, IPEC-J2 cells were seeded onto six-well cell culture plates and maintained in the above mentioned media. The cells were allowed to adhere for 24 hours before being washed and re-fed every other day for seven days to allow for the formation of a model epithelium. Twenty-four hours before experimentation, cells were washed and replacement media was as above but devoid of antibiotics. Experiment 1 included the following treatments: 1) control (CTL; growth media devoid of antibiotics); 2) CTL + LPS (10 ng/mL); 3) CTL + low $ZnSO_4$ (40 µM); and 4) CTL + high $ZnSO_4$ (80 µM). Experiment 2 included the following treatments: 1) CTL; 2) CTL + LPS (10 ng/mL); 3) CTL + low GENT (50 µg/mL); and 4) CTL + high GENT (100 µg/mL). For both experiments, total RNA was harvested at 1.5, 3 and 6 hours following the addition of the respective treatments.

RNA Isolation and Quantitative Real-Time PCR Analysis

Total RNA was extracted and contaminating DNA was removed from all RNA samples. Samples were reconstituted in nuclease-free water and frozen for further analysis. The quality of RNA was assessed by agarose gel electrophoresis and visualization of the 28S and 18S rRNA bands. The quantity of RNA was determined by spectrophotometry (OD 260 nm). Complementary DNA (cDNA) was synthesized from 1.0 µg of RNA. Reverse transcription was carried out using reverse transcription reagents in a 50 µL final volume that included 25 mM $MgCl_2$, 500 µM dNTP's, 2.5 µM random hexamers, 0.4 U/µL Rnase inhibitor, 50 U/µL MultiScribe reverse transcriptase, and TaqMan RT buffer. The reverse transcription mixture was incubated at 25°C for 10 minutes, heated to 37°C for 60 minutes, and inactivated at 95°C for 5 minutes. The resultant cDNA was used as a template for real-time, quantitative polymerase chain reaction (qPCR) in order to quantify ZnT1 mRNA relative to the quantity of the endogenous control (18S rRNA). The qPCR reactions were carried out in 384-well plates with the ZnT1-specific forward and reverse primers and TaqMan TAMRA probe, PCR Mastermix, and 3.5 µL cDNA template. The porcine specific ZnT1 primers and detection probe were synthesized from published GenBank (Accession No. AY918800) sequences using PrimerExpress software. Commercially available eukaryotic 18S rRNA primers and probe were used as an endogenous control. PCR reactions, run in triplicate wells, were carried out with the Applied Biosystems 7900HT Fast Detection System using 40 cycles of amplification with alternating 15 seconds, 95°C denaturation and 1 minute, 60°C anneal/extension cycles.

Statistical Analyses

Relative abundance of ZnT1 mRNA in IPEC-J2 cells were calculated with the ΔΔCT method using the average ΔCT values of cells from control wells as the reference expression. These ΔΔCT values were analyzed using the MIXED procedure (SAS Inst. Inc., Cary, N.C.) to determine the effect of treatment on ZnT1 relative gene expression. The model included effects of treatment, time, and their interaction. Gene expression data from porcine tissues were handled similarly except that the average spleen ΔCT values were used as the reference gene expression (n = tissue from four pigs).

Results and Discussion

In Vivo *Expression of ZnT1 mRNA in Porcine Tissues*

The relative abundance of ZnT1 mRNA in porcine tissues is presented in Figure 1. Expression of ZnT1 mRNA was expressed in all tissues obtained from weaned pigs. In addition, significant differences in ZnT1 mRNA abundance were observed among tissues ($P < 0.001$). The greatest relative abundance of ZnT1 mRNA was observed in the tonsil and was greater ($P < 0.05$) than all other tissues (in

(Continued on next page)

order of decreasing ZnT1 mRNA: thymus, ileum, liver, jejunum, and spleen). This result differs from previous work in humans where ZnT1 mRNA was observed to be most abundant in the duodenum and jejunum. More research is warranted to evaluate ZnT1 mRNA expression in a greater number of animals, over a broader panel of tissues, as well as within different cell types.

In Vitro *Expression of ZnT1 mRNA in IPEC-J2 Cells*

The relative abundance of ZnT1 mRNA from the *in vitro* experiments is presented in Figure 2 ($ZnSO_4$ supplementation) and Figure 3 (GENT supplementation). The relative abundance of ZnT1 mRNA for each of the respective treatments are presented in comparison to CTL cells where the relative abundance of ZnT1 mRNA equals one.

$ZnSO_4$ Supplement on Cultured Media

With respect to experiments where IPEC-J2 cells were supplemented with low and high $ZnSO_4$, no time × treatment interaction was observed. However, ZnT1 mRNA expression tended ($P < 0.1$) to increase over time when means were averaged among all treatments, and ZnT1 mRNA tended ($P < 0.07$) to be increased in response to $ZnSO_4$ supplementation when means were averaged across all time points. These results are in agreement with previous work by others where ZnT1 mRNA in Caco-2 cells (a human colonic epithelial cell line) was increased with supplemental zinc within four hours of exposure.

In the second *in vitro* experiment, GENT supplementation resulted in a significant time × treatment interaction ($P < 0.005$). At 1.5 hours following initial exposure of IPEC-J2 cells to their respective treatments, no effects of treatment were observed on the relative abundance of ZnT1 mRNA. At 3 hours post-treatment, cells treated with high GENT had greater ($P < 0.05$) ZnT1 mRNA compared to all other treaments. At six hours post-treatment, the relative abundance of ZnT1 mRNA was greater ($P < 0.05$) in low and high

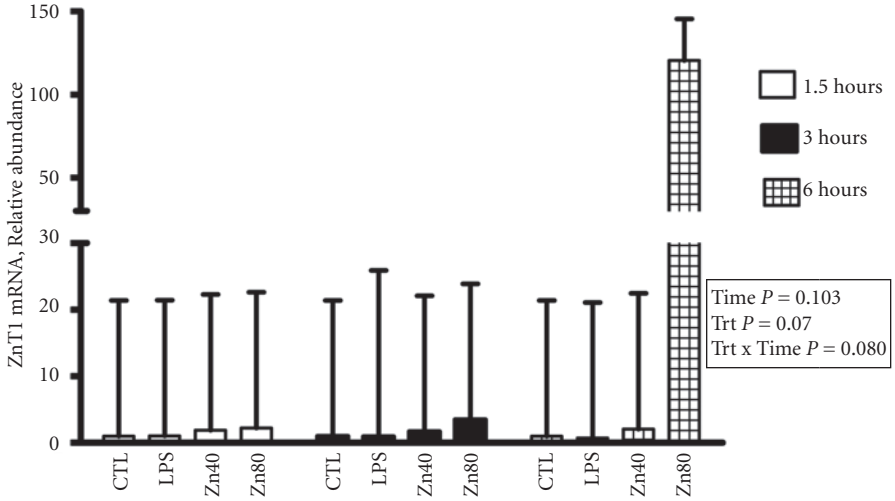

Figure 2. Relative abundance of zinc transporter (ZnT) 1 mRNA from cultured porcine jejunal epithelial cells (IPEC-J2) treated with media alone (CTL), 10 ng/mL lipopolysaccharide (LPS), 40 µM zinc (as zinc sulphate; Zn40), or 80 µM zinc (as zinc sulphate; Zn80). Total RNA extracted at 1.5, 3.0, and 6.0 hours post treatment. Each bar represents the least squares mean (± SEM) of three observations.

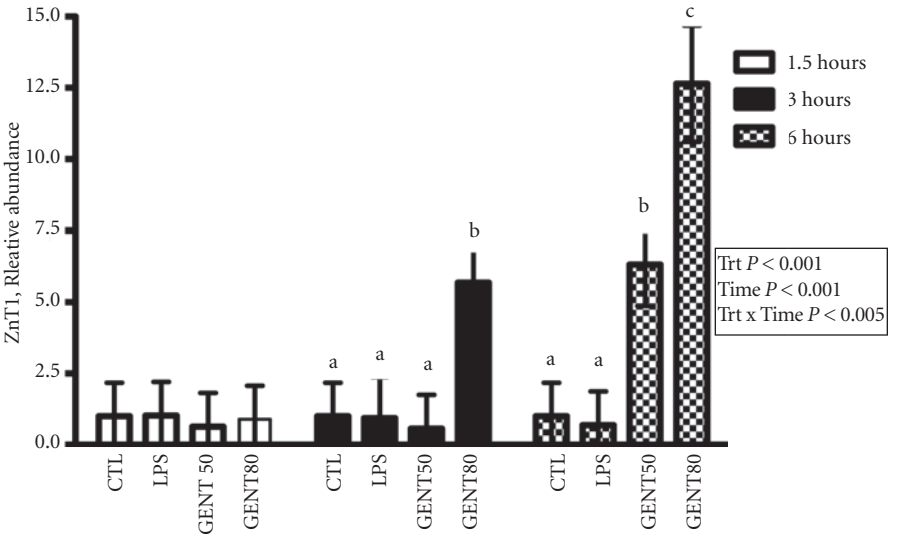

Figure 3. Relative abundance of zinc transporter (ZnT) 1 mRNA from cultured porcine jejunal epithelial cells (IPEC-J2) treated with media alone (CTL), 10 ng/mL lipopolysaccharide (LPS), 50 µM antibiotic (as gentamicin; GENT 50), or 100 µM antibiotic (gentamicin; GENT 100). Total RNA extracted at 1.5, 3, and 6 hours post-treatment. Each bar represents the least squares mean (± SEM) of three observations. Within time periods, bars without common superscripts differ ($P < 0.05$).

GENT treated cells compared to CTL or LPS treated cells. In addition, the relative abundance of ZnT1 mRNA of high GENT treated cells was greater ($P < 0.05$) compared to all other treatments at all time points.

Conclusions

This research indicates that ZnT1 mRNA is differentially expressed in tissues obtained from weaned pigs and that ZnT1 mRNA is differentially regulated in IPEC-J2 cells in response to zinc and antibiotic supplementation. Additional studies are underway to evaluate ZnT1 mRNA expression in pigs and to determine the mechanisms by which zinc supplementation affects animal growth and health.

[1]Thomas E. Burkey is an assistant professor, Phillip S. Miller is a professor, and Huyen Tran is a graduate student in the Animal Science Department at the University of Nebraska–Lincoln.

Effect of Dam Parity on Litter Performance and Passive Immunity

Litter performance and passive immunity may be affected by dam parity.

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Summary

Preliminary experiments (reported in the 2008 Nebraska Swine Report) suggest that progeny health status may be affected by dam parity. However, the preliminary experiments only included a small population of sows and their progeny. Therefore, the objective of the current experiment was to evaluate litter performance and the production and passive transfer of immunoglobulins (Ig) in dams (P1 vs. P4) and their progeny. Litter birth weight tended ($P < 0.10$) to be greater for P4 progeny compared to P1 progeny. No effects of dam parity were observed on circulating Ig in dams during gestation or at parturition. However, concentrations of IgA tended ($P < 0.09$) to be greater for P4 sows compared to P1 gilts in samples of colostrum and milk and serum IgG concentrations were greater ($P < 0.02$) for P4 progeny compared to P1 progeny across all preweaning samples. These results suggest that litter performance and health status may be affected by dam parity.

Introduction

Anecdotal observations suggest that progeny of gilts (P1) have reduced health status and subsequent performance compared to progeny of sows (≥ P2). Unpublished reports demonstrate that P1 progeny have reduced weaning weights, decreased nursery and finishing ADG and greater mortality than P2 progeny. In addition, work by Mahan et al. (1998) showed that mature sows (≥ P2) had a greater litter gain per day than P1 gilts. In all of these reports, it is generally accepted that differences observed between parities result from reduced health status in P1 progeny. Although it remains unclear, several findings may help explain why health status and performance of progeny seems to vary between parities. It is possible that progeny health status is affected by factors including (but not limited to) animal stress, passive immunity, and susceptibility to pathogens. With respect to passive immunity, maternal colostrum provides Ig that are absorbed within the first 24 hours after birth. The concentration of IgG in colostrum ingested by the suckling piglet affects the concentration of circulating IgG in piglet serum. When passive immunity is low or fails, the piglet's health status decreases and may affect survivability. Therefore, receiving adequate colostrum in the first 24 hours after birth is extremely important.

Preliminary data generated at the University of Nebraska *(2008 Nebraska Swine Report)* resulted in several interesting observations. First, at parturition, circulating concentrations of immunoglobulins (IgA and IgG) were greater in P3 dams compared to P1 dams. Second, with respect to immunoglobulin concentrations during lactation, no parity differences were observed between P1 and P3 dams. Third, circulating concentrations of immunoglobulins (IgA and IgG) were greater in P3 progeny compared to P1 progeny in samples obtained at weekly intervals from birth to 37 days of age. These three observations suggest that passive immunity may be affected by dam parity. It is important to emphasize that the aforementioned observations are a result of a preliminary study conducted with a limited number of observations. Therefore, the objective of the current experiment was to evaluate, on a larger scale, litter performance and production and passive transfer of IgG and IgA between P1 and P4 dams and their progeny.

Materials and Methods

Experimental Design

The experimental protocol was reviewed and approved by the Institutional Animal Care and Use Committee of the University of Nebraska–Lincoln. Dams (Large White × Landrace) utilized in the current study included first parity gilts (P1; n = 19) and fourth parity sows (P4; n = 24) that all farrowed over a 22-day period beginning December 17, 2007, and ending January 7, 2008. Dams were co-mingled and housed in stalls during gestation and moved to farrowing crates approximately five days prior to their expected farrowing date. Dam and litter performance was recorded for both P1 and P4 females. The dam and litter performance parameters recorded included: No. of pigs/litter (total born, born live, stillbirths, mummified fetuses, pigs weaned, and preweaning mortality), litter weight at birth (LBW), and litter weight at weaning (LWW). All piglets from each litter were weighed on day 0, 7, 14, and at weaning (day 19).

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Laboratory Analysis

Blood samples were collected from all sows via jugular venipuncture at two time points during gestation (day 90 and 114) and at a final time point immediately following parturition (day 0). During lactation, samples were obtained at day 0 (colostrum), 7 (mid lactation), and 14 (late lactation) from each functional teat in sterile flasks and frozen (-20°C) for subsequent analyses. For mid- and late-lactation milk collection, oxytocin was administered to facilitate milk collection. Colostrum and milk samples were diluted (1:50,000) and concentrations of IgA and IgG were quantified as described below. Blood samples were collected from six piglets from each litter on day 1, 7, and 14. Serum was harvested by centrifugation (20 minutes at 1,500 × *g*) and frozen for subsequent analyses. Concentrations of immunoglobulins (IgA and IgG) in serum, colostrum, and milk were quantified via swine-specific enzyme-linked immunosorbent assays (ELISA; Bethyl Labs Inc., Montogomery, Tex.).

Statistical Analysis

The MIXED procedure of SAS was used to analyze the progeny serum and lactation data as a completely random design with repeated measures over time on each experimental unit. The model included terms for the fixed effects of parity and time and their interaction. Comparisons between parity and time were made only when a significant ($P < 0.05$ unless noted otherwise) F-test for the main effect or interaction was detected using the least significant difference procedure. All means presented are least squares means. Litter performance data was analyzed using the MIXED procedure of SAS as a completely randomized design.

Table 1. Treatment effects of sow parity on litter and pig measurements.

	Parity			
Item	1	4	SEM[a]	*P*-value
No. of sows	19	24		
Pigs, No./litter				
Total born	12.79	12.79	1.2	0.99
Born live	12.00	11.50	1.1	0.66
Stillbirths	0.63	1.13	0.3	0.12
Mummified fetuses	0.16	0.21	0.2	0.68
Mortality (preweaning)	2.68	1.83	0.8	0.26
Weaned	10.16	10.13	0.6	0.96
Litter wt, lb				
Birth (day 0)	15.73	17.89	1.3	0.10
Weaning (day 19)	55.14	58.07	4.8	0.55

[a]SEM = Standard error of the mean

Results and Discussion

Dam and litter performance are presented in Table 1. There was no effect of parity on number (pigs/litter) of total born, born live, stillbirths, mummified fetuses, deaths, or on LWW. However, P4 dams tended to have greater LBW compared to P1 dams ($P = 0.10$) and P4 dams had a numerical decrease in preweaning mortality (number of deaths) and a numerical increase in LWW. Progeny BW is represented in Figure 1. There was no significant parity × time interaction for piglet BW. However, across all time points P4 progeny had greater ($P < 0.0005$) BW than P1 progeny. We expected to have greater differences in dam and litter performance; however, according to previous research it is possible that the greatest differences in performance occur between P1 and P2 or P3 dams.

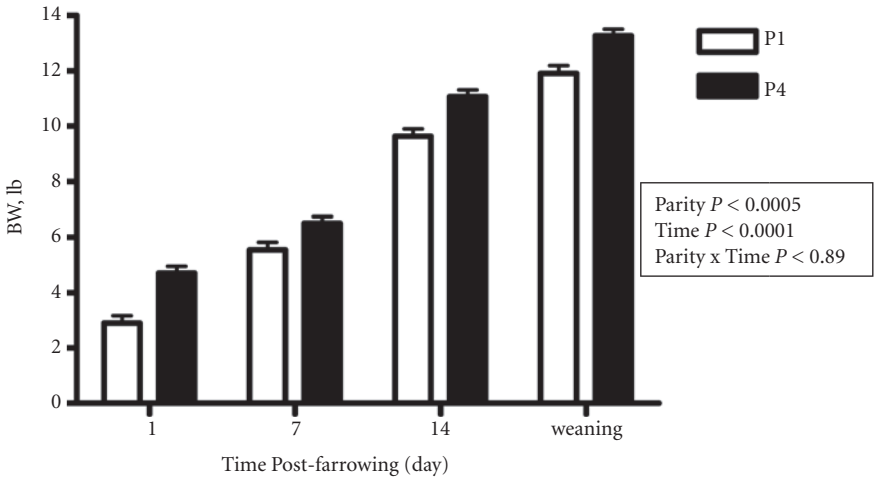

Figure 1. Average body weight (BW) of piglets of gilts (P1) and sows (P4) taken on day 0, 7, and 14 following parturition. Each bar represents the least squares mean (± SEM) of the progeny of 19 and 24 observations for P1 and P4 dams, respectively.

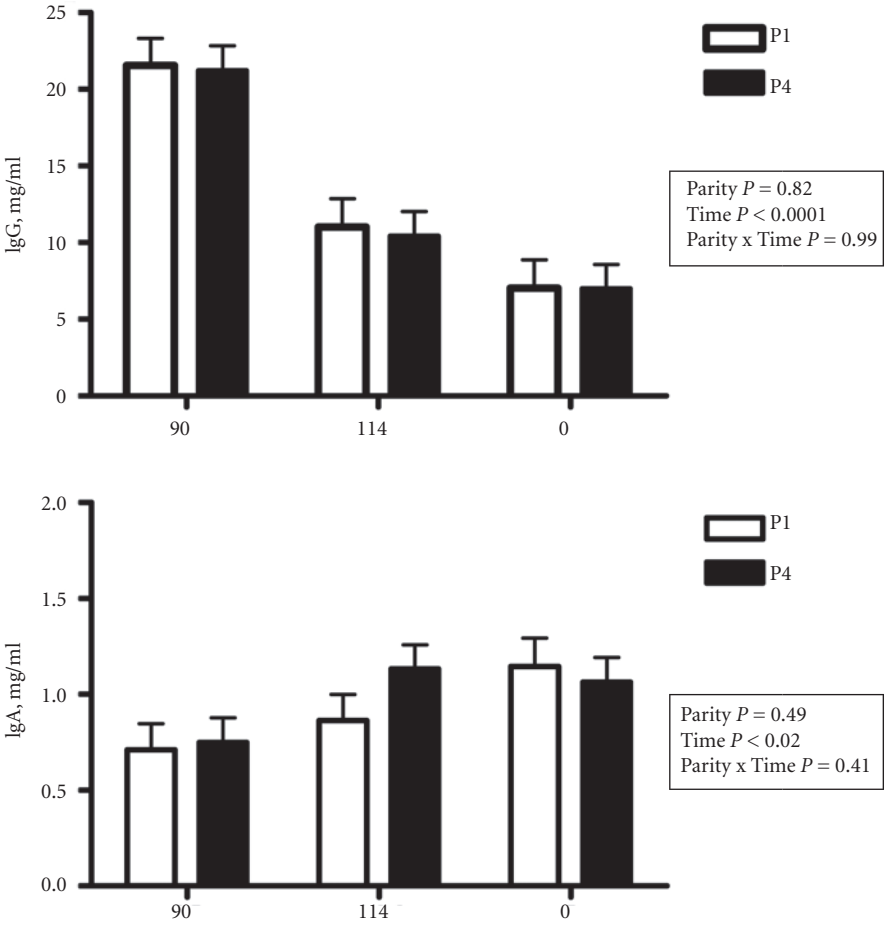

Figure 2. Circulating concentrations of IgG (top panel) and IgA (bottom panel) in gilts (P1) and sows (P4). Immunoglobulin concentrations were evaluated in serum obtained at day 90 and 144 of gestation and immediately following parturition (day 0). Each bar represents the least squares mean (± SEM) of 19 and 24 observations for P1 and P4 dams, respectively.

Figure 2 depicts circulating concentrations of IgG and IgA in P1 and P4 dams during gestation (day 90 and 114) and following parturition (day 0). A significant parity × time interaction was not observed and there were no main effects of parity on circulating Ig. However, consistent with previous reports, there was a significant effect of time when means were averaged across both parities ($P < 0.05$). While circulating IgA increases as the dams approached farrowing, circulating IgG concentrations decreased over time with the lowest concentrations observed at farrowing (day 0). This observation may contribute to the higher levels of IgA in mid- and late-lactation milk when compared to IgG.

Concentrations of IgG and IgA in samples of colostrum and milk obtained during lactation are represented in Figure 3. There was no significant parity × time interaction or main effect of parity on IgG concentrations during lactation. However, IgG concentrations averaged across both parities were greater for colostrum when compared to mid- and late-lactation samples ($P < 0.05$). With respect to IgA, no parity × time interaction was observed. However, there was a tendency for IgA concentrations to be greater in P4 dams compared to P1 dams when means are averaged across all time points ($P = 0.09$). Similar to IgG, the greatest concentrations of IgA were observed during early lactation (colostrum) ($P < 0.05$).

No significant parity × time interactions were observed for either IgG or IgA when circulating Ig concentrations were measured in serum from P1 and P4 progeny (Figure 4). Piglets derived from P4 dams had greater circulating IgG concentration when compared to P1 progeny when means are averaged across all time points ($P < 0.02$). There was no main effect of parity on circulating IgA concentrations in P1 and P4 progeny; however, P1 progeny had numerically decreased IgA concentrations compared to P4 progeny at day 1 and 7.

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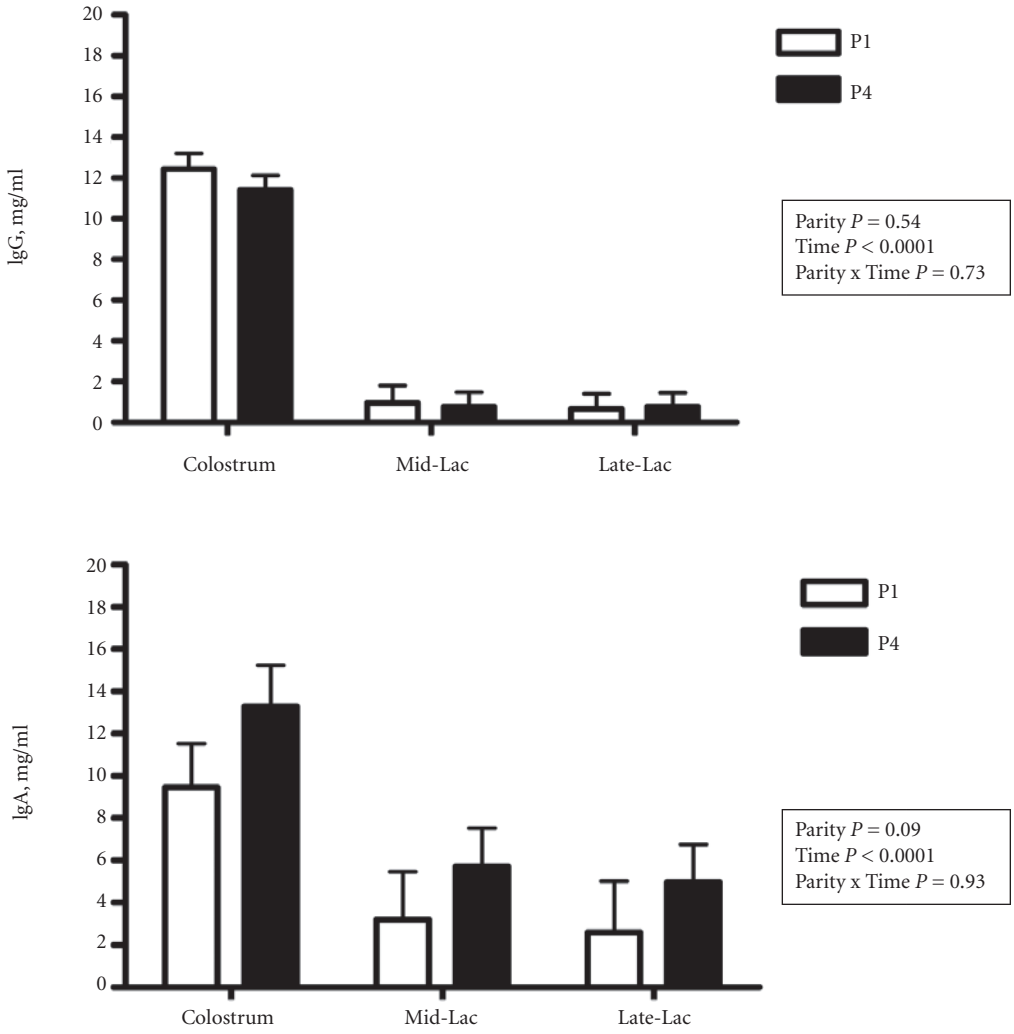

Figure 3. Concentrations of IgG (top panel) and IgA (bottom panel) in colostrum, mid lactation (7 days following parturition; mid-lac), and late-laction (14 days following parturition; Late-Lac) milk samples obtained from gilts (P1) and sows (P4). Each bar represents the least squares mean (± SEM) of 19 and 24 observations for P1 and P4 dams, respectively.

As expected, a main effect of time was observed for both IgG and IgA with circulating Ig concentrations decreasing over time when means are averaged across both parities ($P < 0.001$).

Conclusion

The level of passive immunity in a given population of piglets varies according to the amount of colostrum they ingest. In addition, the level of passive immunity acquired may directly affect the development of active immunity and indirectly affect the health and performance of the piglet. The results described in this report suggest that mature dams (P4) may provide their progeny with advantages in provision of passive immunity. However, further research is needed to determine if these observations are consistent throughout the sow's reproductive lifetime.

[1]Erin E. Carney, Huyen Tran, and Justin W. Bundy are graduate students; Roman Moreno is a graduate student and research technologist; Phillip S. Miller is a professor; and Thomas E. Burkey is an assistant professor in the Animal Science Department at the University of Nebraska–Lincoln. Matthew W. Anderson is manager and Jeffrey M. Perkins is a research technician at the UNL Swine Research Farm.

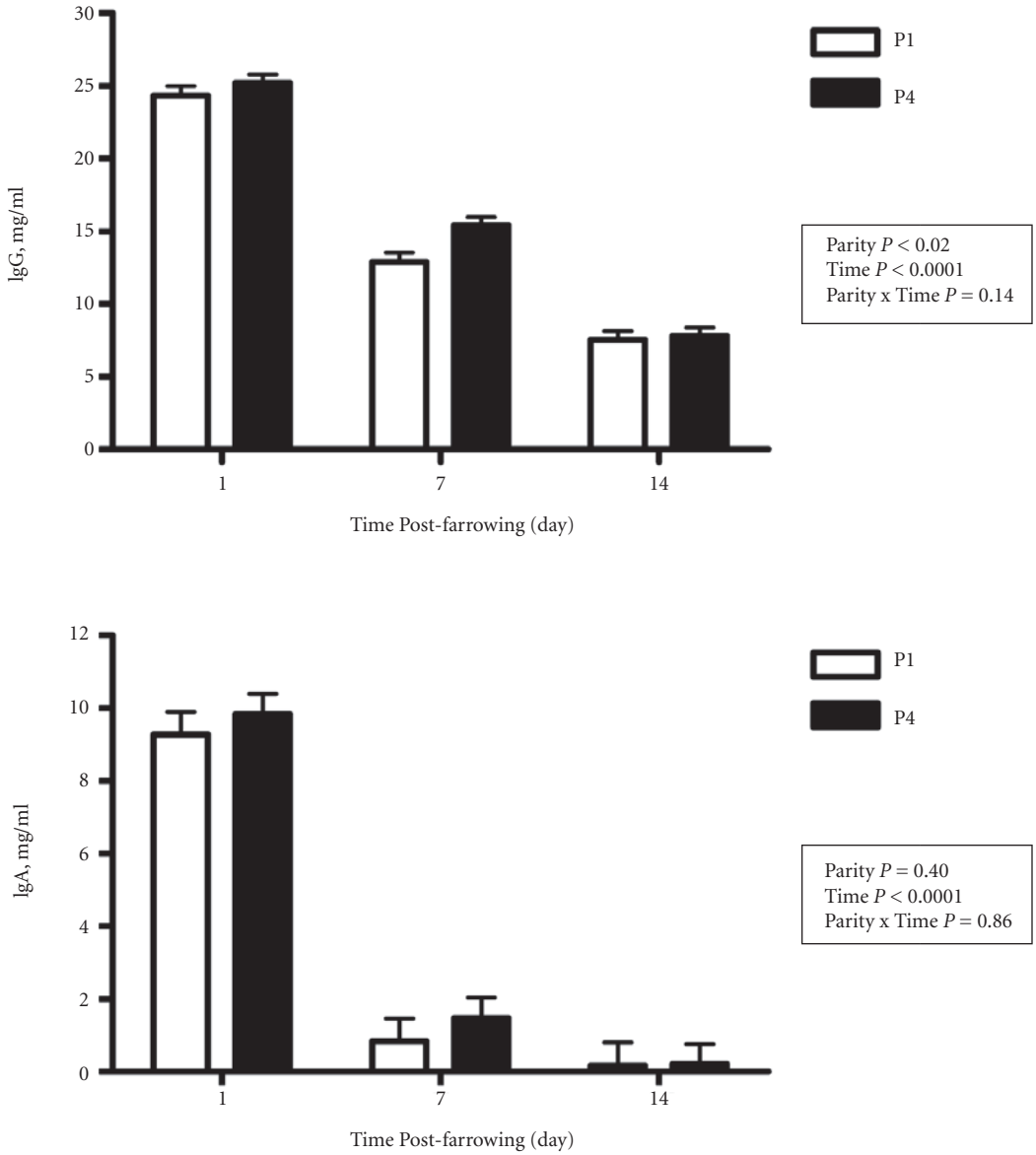

Figure 4. Circulationg concentrations of IgG (top panel) and IgA (bottom panel) in serum obtained from the progeny of gilts (P1) and sows (P4). Immunoglobulin concentrations were evaluated in serum obtained at 1, 7, and 14 days post-farrowing. Each bar represents the least squares mean (± SEM) of the progeny of 19 and 24 observations for P1 and P4 dams, respectively.

Effect of Dam Parity on Growth Performance and Immunity of Weaned Pigs

Growth performance in the nursery may be affected by dam parity.

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Roman Moreno
Phillip S. Miller
Thomas E. Burkey[1]

Summary

The growth performance of weaned pigs derived from different parities has not been previously evaluated; however, unpublished and anecdotal observations suggest that progeny derived from first parity (P1) dams have reduced health status and growth performance compared to progeny derived from mature sows (≥ P2). The objective of the current study was to evaluate the effect of dam parity on growth performance and immune response of P1 and P4 progeny during the nursery phase of production. Results from this experiment suggest that P4 progeny have increased body weight and growth performance during the nursery phase of production compared to P1 progeny. There were no effects of dam parity on immune response in this experiment.

Introduction

Parity segregation is used on some commercial swine farms. Parity 1 progeny are often segregated from the progeny of more mature dams (≥ P2) because of decreased growth performance and higher mortality rates. The effect of dam parity on progeny growth performance is not fully understood. However, it has been generally accepted that P1 progeny have reduced growth performance when compared to progeny of mature dams. This could be due to a lower health status in P1 progeny, causing decreased average daily gain (ADG) and increased mortality. Unpublished data suggest that P1 progeny have reduced weaning weight, decreased nursery and finishing ADG, and greater mortality than P2 progeny. The objective of the current study was to evaluate the effect of dam parity on growth performance and immune response of P1 and P4 progeny during the nursery phase of production.

Materials and Methods

Experimental Design

The experimental protocol was reviewed and approved by the Institutional Animal Care and Use committee of the University of Nebraska–Lincoln. A total of 96 weaned pigs were used in a 42-day study that is a continuation of the experiment described in a previous report (see previous article: "Effect of Dam Parity on Litter Performance and Passive Immunity"). To obtain an accurate representation of body weight (BW) as the pigs were removed from the farrowing house, BW was averaged within parity (P1 or P4), and pigs were selected based on the average BW of each parity. Initial BW of P1 and P4 pigs averaged 12.56 and 13.98 $\pm$ 0.1 lb, respectively.

Six pigs were housed in each pen with four replications per treatment. Pigs within each parity were allotted to one of two dietary treatments, a control diet (CTL) or the CTL diet with the antibiotic Mecadox (50 lb/ton; AB). This created four treatments for the nursery study, consisting of: 1) P1, CTL; 2) P1, AB; 3) P4, CTL; and 4) P4, AB (Table 1). All diets were fed in meal form and formulated to meet or exceed NRC requirements for growth. Pigs were fed in three phases: Phase I (day 0 to 7); Phase II (day 8 to 21); and Phase III (day 22 to 42). Pigs were housed in a temperature-controlled room and each pen contained a single nipple waterer and a single self-feeder to facilitate ad-libitum access to water and feed. Weight and feed disappearance were recorded on day 7, 21, and 42. Average daily gain, average daily feed intake (ADFI), and ADG:ADFI (G:F) were estimated based on the weekly pen BW and feed disappearance.

Table 1. Composition of phase 1, 2, and 3 diets (as-fed basis) %.

	Control			Antibiotics		
Ingredients (%)	Phase 1	Phase 2	Phase 3	Phase 1	Phase 2	Phase 3
Corn	44.4	43.9	60.2	43.4	42.9	59.2
Soybean meal, 47.5% CP	14.8	32.0	33.8	14.8	32.0	33.8
Whey, dried	22.5	15.0	0.0	22.5	15.0	0.0
Fish meal	8.0	4.0	0.0	8.0	4.0	0.0
Animal plasma	6.0	0.0	0.0	6.0	0.0	0.0
Corn oil	3.0	3.0	3.0	3.0	3.0	3.0
Dical phosphate	0.4	1.0	1.7	0.4	1.0	1.7
Limestone	0.3	0.4	0.6	0.3	0.4	0.6
Salt	0.3	0.3	0.3	0.3	0.3	0.3
Swine vit premix	0.3	0.3	0.3	0.3	0.3	0.3
Swine TM premix	0.2	0.2	0.2	0.2	0.2	0.2
L-Lysine HCl	0.0	0.0	0.0	0.0	0.0	0.0
DL-Methionine	0.1	0.0	0.0	0.1	0.0	0.0
Mecadox - 2.5 g/lb	0.0	0.0	0.0	1.0	1.0	1.0

Table 2. Effect of dam parity and dietary treatment on average body weight (BW), average daily gain (ADG), average daily feed intake (ADFI) and feed efficiency (G:F) of weaned pigs.

	Treatments[a]					*P*-values		
Parity	1	1	4	4				
Diet	CTL	AB	CTL	AB	SEM[b]	1[c]	2[d]	3[e]
No. of pigs	24	24	24	24				
BW, lb								
day 0	12.681	2.44	14.10	13.85	0.11	0.0001	0.324	0.991
day 7	14.58	13.93	15.46	15.27	0.12	0.001	0.14	0.4
day 21	24.95	25.07	27.47	26.86	0.30	0.007	0.722	0.588
day 42	55.06	52.01	59.79	54.00	0.70	0.014	0.054	0.867
Phase 1 (day 0 to 7)								
ADG, lb	0.27	0.21	0.19	0.20	0.01	0.185	0.443	0.307
ADFI, lb	0.28	0.28	0.30	0.31	0.01	0.244	0.908	0.636
G:F, lb/lb	0.97	0.76	0.64	0.66	0.11	0.077	0.393	0.334
Phase 2 (day 8 to 21)								
ADG, lb	0.74	0.81	0.86	0.83	0.02	0.145	0.647	0.266
ADFI, lb	0.98	1.05	1.14	1.24	0.02	0.005	0.142	0.804
G:F, lb/lb	0.76	0.78	0.75	0.68	0.04	0.229	0.538	0.282
Phase 3 (day 22 to 42)								
ADG, lb	1.43	1.34	1.54	1.40	0.03	0.217	0.082	0.691
ADFI, lb	2.25	2.21	2.47	2.36	0.03	0.02	0.312	0.611
G:F, lb/lb	0.64	0.61	0.62	0.59	0.02	0.303	0.048	0.964
Overall (day 0 to 42)								
ADG, lb	1.01	0.97	1.09	1.01	0.02	0.092	0.088	0.554
ADFI, lb	1.50	1.49	1.66	1.64	0.02	0.09	0.641	0.878
G:F, lb/lb	0.68	0.65	0.65	0.61	0.02	0.087	0.07	0.588

[a]Dietary treatments included: parity 1 progeny fed the control diet (P1, CTL), parity 1 progeny fed the control diet plus antibiotic (P1, AB), parity 4 progeny fed the control diet (P4, CTL), or parity 4 progeny fed the control diet plus antibiotic (P4, AB).
[b]Standard Error of the Mean.
[c]Main effect of parity [(P1, CTL + P1, AB) vs. (P4, CTL + P4, AB)].
[d]Main effect of dietary treatment [(P1, CTL + P4, CTL) vs. (P1, AB + P4, AB)].
[e]Parity × treatment interaction.

Table 3. Frequency and serum antibody titers of pigs serologically positive for *Mycoplasma hyopneumoniae* (*M. hyo*) by the Tween 20 enzyme-linked immunosorbent assay (ELISA).

Parity	Dietary Treatment	Vaccinate or Saline Control	No. of pigs	Pigs positive (%)[a]	Tween 20 serum antibody titers (mean OD ± SEM)[b]
1	CTL	Saline	8	0	0.081
		Vaccinate	8	8 (100)	0.523
1	AB	Saline	8	0	0.077
		Vaccinate	8	7 (87.5)	0.527
4	CTL	Saline	8	0	0.075
		Vaccinate	8	8 (100)	0.452
4	AB	Saline	8	0	0.078
		Vaccinate	8	8 (100)	0.487

[a]Samples were considered positive if OD ≥ 0.24.
[b]Back-transformed geometric mean titer ± standard error of the mean (SEM).

On the day of weaning (day 0), four pigs (two barrows, two gilts) per pen were randomly chosen and vaccinated against *Mycoplasma hyopneumoniae* (*M. hyo*; RESPISURE[2]). Two pigs (one barrow, one gilt) were given the vaccine and two pigs (one barrow, one gilt) were administered a saline control. All pigs received booster vaccinations (or the appropriate saline control) at day 14 and day 28. Serum was collected from all pigs at the conclusion of the experiment (day 42) and forwarded to the University of Minnesota Veterinary Diagnostic Laboratory to be assayed for antibodies to *M. hyo* by employing the Tween 20 diagnostic assay. Titer values from two pigs (two vaccinates, two saline controls) within pens were averaged to produce a pen mean *M. hyo* titer for statistical analysis.

Statistical Analysis

Each pen was considered an experimental unit. The model was a completely randomized design. Growth data were analyzed using the MIXED procedure of SAS with dietary treatment and parity as the main effects of the model. Pen was considered as a random effect. Pig BW on day 0 was used as a covariate in the statistical analyses.

Results and Discussion

Body weight and growth performance are reported in Table 2. There was no parity × dietary treatment interaction for BW during this experiment. However, BW of P4 progeny were greater ($P < 0.02$) than P1 progeny on day 0, 7, 21, and 42 when means were averaged among dietary treatments. There were no effects of dietary treatment (CTL or AB) on BW on day 0, 7, or 21 when means were averaged among parities; however, on day 42 pigs fed the CTL diet tended to have greater ($P = 0.054$) BW than pigs fed the AB diet.

With respect to growth performance, no significant parity × dietary treatment interaction was observed for ADG, ADFI, or G:F during the entire length of the study. During Phase I, there were no significant main effects of parity or dietary treatment on ADG or ADFI; however, G:F tended ($P = 0.08$) to increase for P1 progeny compared to P4 progeny. During Phase II, there were no main effects of parity or dietary treatment on ADG or G:F; however, ADFI was greater ($P < 0.005$) for P4 progeny compared to P1 progeny. During Phase III, P4 progeny had greater ADFI compared to P1 progeny ($P < 0.02$), and pigs fed the CTL diet had greater ($P < 0.05$) G:F and tended ($P = 0.08$) to have greater ADG than pigs fed the AB diet.

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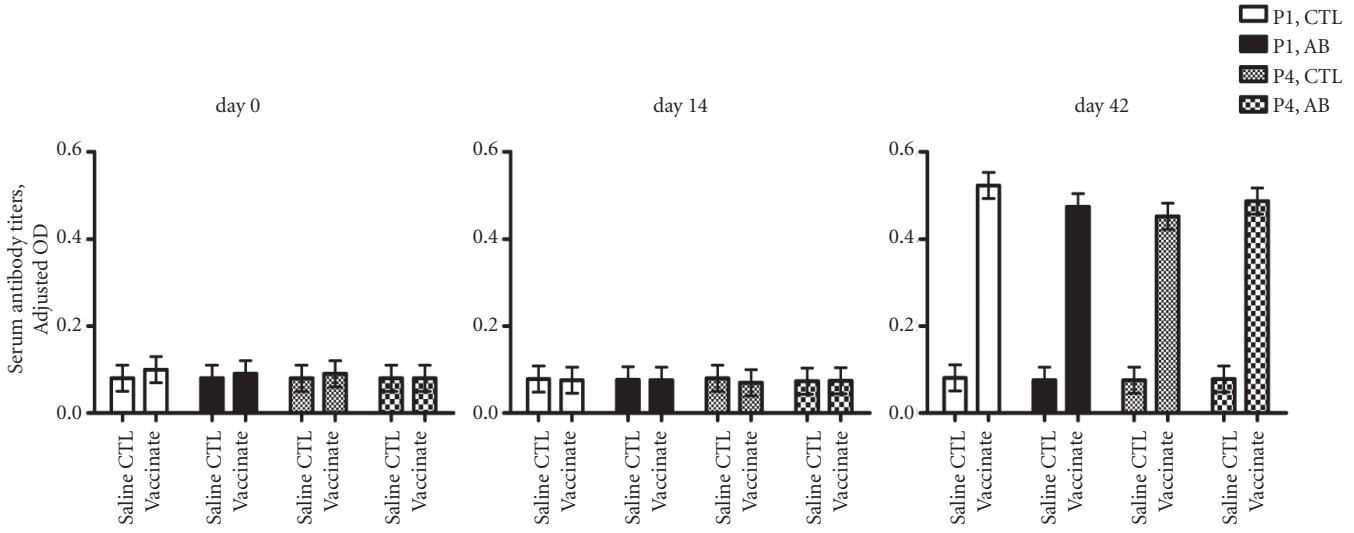

Figure 1. Mean serum antibody titers for *Mycoplasma hyopneumoniae* (*M. hyo*) by the Tween 20 enzyme-linked immunosorbent assay (ELISA) on day 0, 14 and 42 of the experiment. Each bar represents the back-transformed geometric mean titer expressed as optical density (OD) ± standard error of the mean (SEM). Dietary treatments included: parity 1 progeny fed the control diet (P1, CTL), parity 1 progeny fed the control diet plus antibiotic (P1, AB), parity 4 progeny fed the control diet (P4, CTL), or parity 4 progeny fed the control diet plus antibiotic (P4, AB).

There was no main effect of antibiotic on ADFI. Overall (day 0 to 42), P4 progeny tended ($P = 0.09$) to have greater ADG and ADFI, and decreased G:F compared to P1 progeny. In addition, pigs fed the CTL diet tended to have greater ADG ($P = 0.09$) and G:F ($P = 0.07$) compared to pigs fed the AB diet. The trend for increased growth performance in P4 progeny is consistent with previous, unpublished reports; however, the tendency for AB fed pigs to have decreased growth performance is surprising. The effect (or lack thereof) of AB on growth performance in the current study may be explained by the overall high health status of the pigs and that these pigs had been reared in an environment where an antibiotic response is not typically detected.

Mean serum antibody titers for *M. hyo* are presented in Figure 1. No pigs were serologically positive for *M. Hyo* antibody titers on day 0 or 14 of the experiment. As expected, all pigs vaccinated against *M. hyo* (with the exception of one pig) were seropositive on day 42.

The frequency and serum antibody titers of pigs serologically positive for *M. hyo* on day 42 of the experiment are presented in Table 3. There were no effects of parity, dietary treatment, or their interaction on *M. hyo* titers. These data are presented with the caveat that titers to *M. hyo* is only one means by which an immune response may be measured. Many factors may contribute to overall health status and more research may be warranted to adequately gauge immune response and overall health status among progeny derived from different parities.

Conclusion

Dam parity may affect growth performance of weaned pigs throughout the nursery period. Additional research is needed to determine the effects of dam parity on animal health in the nursery and on growth performance through the growing and finisher phases of production.

[1]Erin E. Carney, Huyen Tran, and Justin W. Bundy are graduate students; Roman Moreno is a graduate student and research technologist; Phillip S. Miller is a professor; and Thomas E. Burkey is an assistant professor in the Animal Science Department at the University of Nebraska–Lincoln.

[2] The authors wish to thank Pfizer Animal Health for providing Respisure and for subsequent analytical analyses.

Selection for Immune Responses to Porcine Circovirus (PCV2) to Decrease Incidence of Porcine Circovirus Associated Disease (PCVAD)

Selection for 65-day weight along with PCV2 viremia and antibody titers offers great potential to decrease incidence of PCVAD.

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Roman Moreno
Alan R. Doster
Rodger K. Johnson[1]

Summary

Genetic and environmental effects on incidence of Porcine Circovirus Associated Disease (PCVAD) and immune responses to Porcine Circovirus 2 (PCV2), and their relationships with body weights were studied in 3,440 pigs of the Nebraska litter size selection lines. Pigs were weighed at birth, weaning, 65, and 180 days of age and scored for symptoms of PCVAD every 10 days from 70 to 180 days of age. Necropsies were performed to confirm accuracy of scoring. PCV2 viremia, and antibodies to PCV2, Porcine Reproductive and Respiratory Syndrome Virus, Mycoplasma hyopneumoniae and Actinobacillus pleuropneumoniae were measured in serum from blood samples drawn at various ages from live pigs and in tissues of pigs necropsied. PCV2b genotype was confirmed to be the pathogen causing PCVAD; other pathogens studied were not involved. Pigs with no symptoms of PCVAD had significantly greater weights (0.22, 1.12, 6.6, and 46.0 lb, at birth, weaning, 60 d, and 180 d, respectively) than affected pigs. Heritability of PCVAD score was 16% ± 4%. The location in which pigs were raised accounted for 22% of the variation in PCVAD score. Nearly all pigs were non-viremic until 90 days of age, but many had antibody titers at weaning and at 60 days of age. These maternal antibodies appeared to protect pigs from PCVAD until approximately 90 days of age. Heritability of viremia level at 90 days of age was greater than at 125 days of age (38 ± 11% vs 11 ± 8%). Genetic variation existed for antibody titers at 90 (h^2 = 55 ± 21%) and 125 days of age (h^2 = 10 ± 8%). Incidence of PCVAD was correlated genetically with body weights, PCV2 viremia level, PCV2 antibody titers, and body weights. Expected response to direct selection for reduced PCVAD score was very low (-0.89% in one generation), whereas expected response to index selection for 65-day weight and PCV2 viremia and antibody titers at 90 days of age was -8.0%, 998% greater than direct selection. Genomic selection for decreased incidence of PCVAD is feasible.

Introduction

Porcine Circovirus Associated Disease (PCVAD), caused by Porcine Circovirus 2 (PCV2), causes high economic losses to pork producers. Symptoms of PCVAD in the University of Nebraska–Lincoln swine research herd were first observed in 2002. Not all pigs on the farm were infected and the incidence rate varied depending on the genetic makeup of the pigs, their location at the farm, and season of the year. The incidence rate in crossbred pigs was very low, but a significant number of pigs of the UNL lines selected for increased litter size were affected. Some pigs seemed to be highly sensitive to the disease whereas others in the same pen remained healthy and showed no symptoms. Usually, only one or two pigs in a pen were affected, but it was common for a high percentage of pigs within some litters to be affected, even when raised in different locations.

These observations pointed to underlying genetic variation in the immune response of pigs to the PCV2 virus. A study of PCVAD conducted at another institution supports this hypothesis as the incidence rate was greater in some breed crosses than others. However, sample size in that experiment was too small to determine the degree of genetic variation (heritability) in incidence rate of PCVAD and in immune responses to PCV2 virus. If sufficient genetic variation exists, then greater resistance to PCV2 and reduced incidence of PCVAD through selection are possible. When practiced in nucleus breeding populations, greater resistance achieved through selection can be transmitted through the breeding pyramid to commercial producers, possibly reducing the need for vaccination.

We therefore conducted a study in which pigs were systematically scored for PCVAD, weighed and bled to create a database for genetic analyses. Because secondary pathogens are often thought to be involved in expression of PCVAD, pigs were also characterized for Porcine Reproductive and Respiratory Syndrome Virus (PRRVS), *Mycoplasma hyopneumoniae* and *Actinobacillus pleuropneumoniae* to determine whether these pathogens were involved along with PCV2 in expression of PCVAD.

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Population

Pigs were from generations 24 to 26 of UNL selection lines, Lines 2 and 45, and control lines, Lines 16 and 61, that were derived from a Large White-Landrace composite population formed in 1979. Lines 2 and 45 have been selected for increased litter size (generations 1 to 20) and increased litter size, increased growth, and decreased backfat, generations 21 to 27. Lines 16 and 61 were randomly selected. Each line is maintained with 40 to 45 litters per generation. Line 2 and 16 litters were born in July and August (denoted contemporary group 1, CG1) and Line 45 and 61 litters were born in January and February (CG2).

Pig Management, PCVAD Scoring, and Serum Sampling

Within two days of farrowing, fostering of pigs among sows both within and between lines was practiced. Fostering could not be accomplished uniformly in all litters. Total number and number of live pigs per litter, pig birth (n = 3,440) and weaning weights (n = 3,438), and number after foster and weaned per foster dam (NW) were recorded.

Pigs were weaned at approximately 17 days and grouped by age in nursery pens of 30 pigs per pen. At 60-65 days of age, four boars from each of the 15 largest litters and four to five females from each of the largest 20 litters in Lines 2 and 45 were identified as candidates for selection as breeders. Two sons per sire and one to two gilts per litter were randomly selected as candidates for breeding in Lines 16 and 61. Final selections occurred at 180 days of age.

All breeder candidates were placed in one of six rooms, eight pens per room and 10 pigs per pen, in a confined, mechanically ventilated building denoted as Location 1. Remaining pigs were placed in one of three locations. Location 2 was a confined building, 25 pens of 10 pigs per pen, with natural ventilation regulated by thermostatically controlled curtains over windows. Location 3 was a confined building, 23 pens of 10 pigs per pen, with natural ventilation controlled manually by adjusting doors over windows. Location 4 was five outdoor lots containing a small hoop structure with straw bedding. There were 50 to 60 pigs per lot. Pigs in CG1 were in Locations 1, 2, and 3; pigs in CG2 were in Locations 1, 2, and 4.

Pigs with symptoms of PCVAD were first observed in 2002 (generation 21). Observations in subsequent years led us to a protocol that produced data for genetic analyses. During Generations 24, 25, and 26 pigs were systematically weighed, scored for PCVAD, and blood samples were drawn. Weight at 65 days was recorded for 2,646 pigs (some pigs were not weighed) when they were placed in finishing pens and weight at 180 days (n = 3,115) were recorded. Beginning 7 days after pigs were placed in finishing pens, they were scored for symptoms of PCVAD once every 7 to 10 days, approximately 10 scores per pig, until 180-day weight was recorded. Pigs with no symptoms received a score of 0, pig with minor symptoms a score of 1, and pigs with definitive symptoms a score of 2. Scores were based on degrees of muscle wasting, growth retardation, rough hair coat, diarrhea, and respiratory distress. A score of 1 was used only to identify pigs for more careful future observation. Only pigs receiving one or more score of 2 were considered positive for PCVAD.

Blood was collected at 60, 90, and 125 days of age from all pigs from Generation 25, CG2, and Generation 26, CGs 1 and 2. Blood was collected from pigs at weaning in Generation 25, CG2 and from sows in Generation 26, CG1 when their pigs were weaned. PCV2 viremia, a measure of the pigs ability to replicate virus, was measured in serum of all pigs with PCVAD score of 2, in serum from a randomly selected pen mate with scores of only 0, in samples of a full sib from another pen, and in samples from two pigs drawn randomly from each birth litter in which no pigs were positive for PCVAD. Serum was sent to Iowa State University Veterinary Diagnostic Laboratory, Ames, Iowa, where Porcine Circovirus II C-ELISA PCR and PCV2 Quantitation (qPCR) were performed to obtain PCV2 viremia and antibody levels.

ELISA was used to test subsamples of serum for M*ycoplasma hyopneumoniae* (MH), *Actinobacillus pleuropneumoniae* (APP), and Porcine Reproductive and Respiratory Virus (PRRSV). Samples collected at 90 (n = 261) and 125 days (n = 228) from pigs from generation 25, CG2, were tested for MH; samples collected at 125 days of age from generation 25, CG2 (n = 228) and Generation 26, CG1 and CG2 (n = 511) were tested for APP. The UNL swine herd is free of PRRSV. To confirm this status, serum from 52 pigs from generations 24 to 26 was tested for PRRSV.

Necropsies were performed on samples of pigs with a PCVAD score of 2 from generation 24, CG1 (n = 10) and CG2 (n = 11), and generation 25, CG2 (n = 17) and in 11 randomly selected pigs with PCVAD score of 0 from generation 25, CG2. Pigs were from all locations and only one pig from any one litter was selected for necropsy. Immunohistochemistry and RT-PCR for PCV2 were performed in lung, cervical lymph node, mesenteric lymph node, tonsil, kidney, and ileum of these pigs. Nasal swabs for RT-PCR testing were collected from five pigs; two of these pigs had no lesions suggestive of PCVAD. Necropsies and RT-PCR were done at the Veterinary Diagnostic Center of the University of Nebraska Department of Veterinary and Biomedical Sciences. These pigs were also tested for PRRSV antibodies by ELISA. Serum from three pigs that were necropsied and that were diagnosed with PCVAD were submitted to the Veterinary Diagnostic Laboratory at Iowa State University, Ames, Iowa where Porcine Circovirus II C-ELISA PCR-PCV2 Quantitation in which the virus was sequenced to determine the specific PCV2 transcript in this herd.

Statistical Procedures

Data were analyzed with procedures appropriate for genetic analyses. Two traits had binomial distributions as the outcome was either yes or no, coded as 0 or 1. These were PCVAD score (1 = positive, 0 = negative) and

Table 1. Number of observations for each trait by generation and contemporary group.

Generation	Group[2]	Trait[1]				
		BWT	WWT	W65	W180	PCVAD score
24	1	669	669	—	659	669
24	2	649	649	543	622	649
25	1	281	281	281	262	281
25	2	511	510	510	431	511
26	1	629	629	624	507	629
26	2	701	698	688	634	701
		Vsows	VW	V60	V90	V125
25	2	—	279	287	261	228
26	1	77	—	292	294	244
26	2	—	—	—	217	211
		IgGsows	IgGWIg	G60Ig	G90Ig	G125
25	2	—	280	287	271	229
26	1	75	—	301	294	244
26	2	—	—	211	217	210

[1]BWT = birth weight; WWT = weaning weight; W65 = weight at 65 d; W180 = weight at 180 d; Vsows = sow viremia at weaning; VW = pig viremia at weaning; V60, IgG60, V90, IGg90, V125 and IgG125 = PCV2 viermia and antibody, respectively, at 60, 90, and 125 days of age.
[2]Group1 = Lines 45 and 61 (CG2, winter litters), Group 2 = lines 1 and 2 (CG1, summer litters).

Table 2. Distribution of pigs with combinations of PCVAD score, virema, and IgG levels at days 90 and 125 days within generation (G) and contemporary group (CG) and overall.

	PCVAD score[1]	Viremic[2]	IgG[3]	G25CG1	G26CG1	G26CG2Total
Day 90						
1	+	+	11	16	0	27
1	+	S	48	64	0	112
1	+	-	27	44	0	71
1	-	+	4	4	14	22
1	-	S	11	3	8	22
1	-	-	7	0	3	10
0	+	+	36	59	1	96
0	+	S	55	68	0	123
0	+	-	18	21	1	40
0	-	+	15	10	103	128
0	-	S	19	5	57	81
0	-	-	11	0	30	41
Day 125						
1	+	+	59	80	9	148
1	+	S	8	3	3	14
1	+	-	3	0	0	3
1	-	+	12	2	0	14
1	-	S	1	0	1	2
1	-	-	0	0	4	4
0	+	+	104	145	78	327
0	+	S	12	3	17	32
0	+	-	0	0	12	12
0	-	+	29	11	13	53
0	-	S	0	0	19	19
0	-	-	0	0	55	55

[1]0 = negative and 1 = positive score for PCVAD.
[2]+ indicates PCV2 viremia level > 0, - indicates PCV2 viremia = 0.
[3]+ = IgG titer ≥ 0.5, S = 0.2 ≤ titer < 0.5, - = titer < 0.2.

whether a pig was viremic (0 = no viral replication, 1 = viral replication, veremia level greater than 0). For those pigs that were viremic (coded score of 1) the observed viremia (genomic copies per ml) were expressed as $\log_{10}$ to normalize the distribution. PCV2 antibody titers and body weights were normally distributed.

Table 1 contains a description of traits and numbers of records. Table 2 contains the joint distributions of PCVAD scores, PCV2 viremia, and PCV2 antibody titers. Genetic analyses used a pedigree file containing 12,032 pigs, all those with phenotypes in the present study and all parent animals tracing back to the base generation. Traits analyzed were PCVAD Score, birth weight, weaning weight, 65-day weight, 180-day weight, viremia scores at 90 days and 125 of age (the 0 or 1 code), $\log_{10}$ of viremia level at 90 days and 125 days of age in pigs with positive viremia scores, and PCV2 antibody titers at 60, 90, and 125 days of age. Antibody titers .5 and greater are considered to be positive, evidence that the pig had been exposed to virus, titers of .2 to .49 are in the suspect range, and those below .2 are considered to be negative.

Results

Observations and Fixed Effects

Overall, 14.4% of pigs had at least one positive PCVAD score, but the incidence varied greatly across generations (Figure 1). Mortality rate of pigs with positive score was 35.4%. Genetic lines did not differ significantly in incidence of PCVAD.

Nearly all serum samples collected at 60 days of age (94.6 %) were negative for PCV2 viremia; therefore viremia at 60 days of age was not analyzed. All but two serum samples collected at 90 days of age from pigs in generation 26, CG2 had negative viremia (Table 2); therefore, data from that group were deleted from analyses of 90-day viremia level. All but two serum samples collected from weaned pigs were negative for PCV2 viremia, but 37 of 77 of their dams were positive. Average antibody titers for sows and

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progeny were 1.07 ± 0.15 and 0.90 ± 0.16, respectively.

Antibody titers for PCV2 at 60, 90, and 125 days of age for each contemporary group are shown in Figure 2. Titers at 60 and 90 days were similar in generation 25, CG2, and generation 26, CG1, greatest in Generation 26.

Frequency of viremic pigs varied greatly across generations, contemporary groups, and ages (Figure 3). In generation 26, CG2, 53.9% of the pigs were non-viremic at 90 days of age but had antibody titers greater than 0.5 (Table 2). Only 5.9% of the pigs in other groups were non-viremic and had high antibody titers. Non-viremic pigs at 125 days of age with antibody titers less than 0.2 occurred in 60.8% of pigs from generation 26 CG2, but in only 10.2% of the pigs in the other groups.

Incidence of PCVAD was greater in males than females and males had greater ($P < 0.05$) PCV2 antibody titers at 60 (0.03) and 90 days (0.05). The probability of being viremic at 90 days of age was less for females than males (-0.30, $P < 0.05$).

Pigs with 0 PCVAD score weighed more ($P < 0.0001$) at birth, weaning, 65 d, and 180 days (0.22, 1.12, 6.6, and 46.0 lb, respectively) than pigs with score of 1. They also had lower PCV2 viremia levels at 90 (0.26 ±0.15) and 125 days (0.85 ± 0.12) and greater antibody titers (0.04 ± 0.01 and 0.05 ± 0.02, respectively).

PCV2 Sequence

All three pigs whose PCV2 mRNA was sequenced were positive for PCV2b genotype. The sequence for one pig was 100% identical to a PCV2 isolate previously characterized and described in the National Center for Biotechnology Information database. The sequences for the other two pigs were not 100% identical to any sequence in the database. One of them had a single base change at position 116 (G to A); the other one had two base changes, one at position 116 (G to A) and one at position 465 (C to G). These findings confirm that PCV2b was the causative virus for PCVAD in this population but that mutations had occurred causing slightly different nucleotide sequences from those previously characterized.

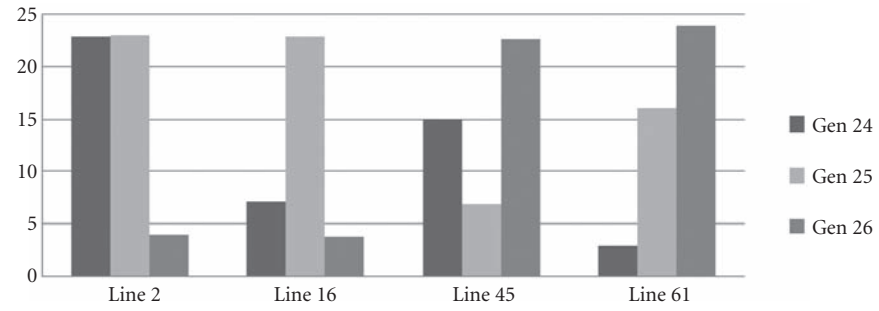

Figure 1. Percentage of pigs scored positive for PCVAD.

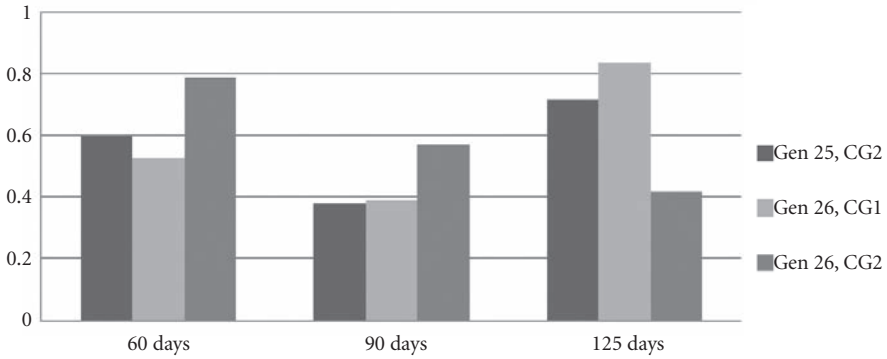

Figure 2. Mean PCV2 antibody titers.

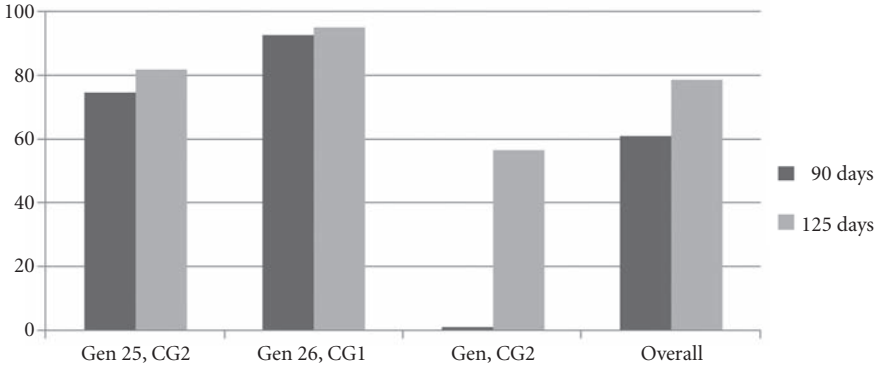

Figure 3. Viremic pigs, %, at 90 and 125 days.

Necropsy Findings.

Tissue samples from 36 of the 38 pigs that were positive for PCVAD were also positive for PCV2. Tissues of the other two were negative, but their nasal swabs were positive. Tissues from all 11 pigs scored as negative for PCVAD were negative for PCV2. Pigs with PCVAD had severe wasting, weight loss, rough hair coat, enlarged mesenteric lymph nodes, lesions indicative of pneumonia, and chronic colitis. Lymphocyte depletion within mesenteric lymph nodes and giant cells in lymphoid follicles, splenic follicles, and Peyer's Patches, and thymic atrophy due to lymphocyte depletion were observed. Nineteen pigs had symptoms of *Mycoplasma hyopneumoniae*; 18 had symptoms of

Table 3. Percentage of variation in PCVAD score and body weights due to different sources.

Source[1]	PCVAD score	BWT, lb	WWT, lb	65-day wt, lb	180-day wt, lb
		Genetic effects			
Pigs genes (h_d^2)	16.0± 4.0	26.7 ±7.0	16.0 ±6.0	23.0 ± 6.02	3.0 ± 6.0
Dam's genes (h_m^2)		18.3			11.0
		Environmental effects			
Birth litter	10.0	11.0			
Weaning litter	27.5	12.0			
Contemporary group	11.2	1.0	0.2	8.0	7.0
Location	22.0	3.0			
Pen	5.1	5.0			
Residual	47.2	43.3	57.1	47.0	51.0
Phenotypic standard deviation	1.46	0.11	0.43	1.97	6.4

[1]h_d^2 = direct heritability, h_m^2 = maternal heritability (± standard error of estimate of h_d^2).

Table 4. Percentage of variance of PCV2 viremia score[1], vermia level[2], and antibody titers[3] at 90 and 125 days of age.[1]

Parameter[4]	VS90	VS125	Viremia90	Viremia125	IgG90	IgG90[2]	IgG125
Pigs genes (h_d^2)			38 ± 11	11 ± 8		55 ± 21	10 ± 8
Birth litter	5.0	4.9			33.3	7.5	5.6
Contemporary group	77.0			18.0	16.7		18.9
Location	2.0	24.0		2.1			33.3
Room		26.2	1.9				6.7
Residual	15.0	44.9	60.2	68.4	50.0	37.5	25.6
σ_p^2	2.57	1.50	1.5	0.97	0.24	0.2	0.3

[1]VS90 and VS125 = viremia score (positive vs 0).
[2]Viremia90 and Viremia125 = $\log_{10}$ (PCV2 genomic copies/mL) for pigs with positive scores.
[3]IgG90 and IgG125 = PCV2 antibody titer.
[2]Generation 26, CG2 data deleted.
[4]h_d^2 = direct heritability ± standard error.

Streptococcus suis, and four had symptoms of *Lawsonia intracellularis.*

ELISA Screening

All serum samples tested for PRRSV and for *Mycoplasma hyopneumoniae* were negative. Thirty two of the 228 serum samples collected at 125 days of age from generation 25, CG2 pigs were positive for APP. Twenty one of 513 serum samples collected at 90 days of age from pigs of generation 26 were positive for APP, 16 had titers in the suspect range, and the rest were negative. Eight pigs that were positive for APP and five with titers in the suspect range had positive PCVAD scores. However, 142 pigs with positive PCVAD scores were negative for APP. Thus, PRRSV and M*ycoplasma hyopneumoniae* can be ruled out as secondary pathogens, and APP was not likely a secondary pathogen, involved in expression of PCVAD in this population.

Genetic and Environmental Parameters

Percentages of total variation due to genetic and environmental effects for PCVAD score and body weights are in Table 3 and those for PCV2 viremia and antibody titers are in Table 4. Direct heritability, a measure of the relative importance of genes of the pig, of PCVAD score was 16 ± 4%. Heritabilities of body weights ranged from 16% for weaning weight to 27% for birth weight. Genes of the dam, maternal heritability, were important for birth and 180-day weights. The location in which pigs were raised accounted for the most variation (22%) in PCVAD score whereas several environmental sources of variation contributed to variation in body weights. For pigs with positive viremia score (levels greater than zero), heritability estimate of viremia level at 90 days of age was greater than at 125 days of age (38 ± 11% vs 11 ± 8%). Genetic variation existed for antibody titers at 125 days of age (h^2 = 10 ± 8%), but not at 90 days of age when all data were included in analyses. However, when data for generation 26, CG2 were deleted, genetic variation in PCV2 antibody titer at 90 days of age was high (h^2 = 55 ± 21%). A large percentage of the variation in viremia score at 90 and 125 days of age was due to either the location or the room in which pigs were raised. These sources of variation were relatively small for viremia level and antibody titers.

Genetic and residual correlations among traits were calculated but are not presented here. The important ones were that PCVAD score was quite highly correlated genetically with day 90 viremia (r_g = 0.75) and antibody level (r_g = -0.67) and moderately correlated with 65-day weight (r_g = -0.53). Neither birth weight nor weaning weight were significantly correlated genetically with viremia or antibody levels. However, weaning weight was highly correlated genetically with viremia at 125 days of age (r_g = -0.73) and 180-day weight was negatively correlated with viremia at both 90 and 125 days of age and positively correlated with antibody titers at 90 days of age. Viremia level at 90 and 125 days of age were positively correlated genetically (r_g = 0.59) and viremia and antibody titers at 90 days of age were negatively correlated (r_g = -0.51). Antibody titers at 90 and 125 days of age were positively correlated (r_g = 0.59).

Although several environmental correlations among traits were significant, none were especially strong. Most notable were that environmental effects on score for PCVAD and for body weights through 65 days of age were correlated ($-0.47 \leq r_e \leq -0.39$), environmental effects on viremia and antibody levels at 90 days of age were correlated (r_e = -0.38), and viremia at 90 days of age was negatively correlated with body weights ($-0.26 \leq r_e \leq -0.16$).

Discussion

We conclude from this study and others in the literature that genetic variation in incidence of PCVAD

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and measures of immune responses to PCV2 exists. The heritability of PCVAD score was 16% and heritabilities of PCV2 viremia and antibody levels at 90 days of age were 38% and 55%, respectively. Genetic variation in viremia and antibody levels at 125 days of age were less and were not significant, perhaps because death of pigs with PCVAD between 90 and 125 days resulted in fewer records and decreased the genetic variance in remaining pigs.

Viremia and antibody levels at 90 days of age and 65-day weight were quite highly correlated genetically with PCVAD score. Thus selection for these traits may be an effective way to decrease incidence of PCVAD. Expected selection responses were calculated for different selection strategies. These were 1) direct selection — selection of breeders only from pigs that never displayed symptoms of PCVAD, 2) single-trait selection for weight, viremia, or antibody levels, and 3) index selection for correlated traits or PCVAD score plus correlated traits.

Even though PCVAD score is heritable, direct selection was relatively ineffective, resulting in a reduction of incidence of PCVAD of only -.8% in the first generation. This result occurred because 85% of the pigs, those with score of 0, were candidates for selection, resulting in a very low selection rate. The other traits are continuously distributed so pigs vary across the entire range of the distribution. Assuming 10% of the males and 30% of the females are selected for high PCV2 antibody titers at 90 days resulted in the greatest correlated expected response to single-trait selection (generation 1 response = -6.5%). Greatest expected response was for a three-trait index including 65-day weight and PCV2 viremia and antibody titers at 90 days of age (-8.0%). A four-trait index of these traits and PCVAD score produced the same expected response per generation. Expected selection responses were 414 to 998% greater when correlated traits were used than from direct selection.

Necropsy results confirmed that PCV2 was the main causative agent of PCVAD in this population. Genotyping PCV2 revealed that PCV2b was likely the causative agent. However, at least three allelic forms, and possibly more, existed. Thus, mutations have occurred in the PCV2b genome, but the consequence is not known.

Porcine Reproductive and Respiratory Virus, M*ycoplasma hyopneumoniae*, and *Actinobacillus pleuropneumoniae* were not secondary pathogens involved in expression of PCVAD. In other work, co-infection of pigs with PCV2 and Gram-negative bacteria induced viral replication of PCV2. Diagnostics for these pathogens were not performed, but infection of pigs with pathogens such as *Escherichia coli* and *Haemophilus influenzae*, Gram-negative bacteria known to be present in this population, may have increased the risk of PCVAD.

A strong relationship between PCV2 viremia level at 90 days of age and incidence of PCVAD across contemporary groups existed. Incidence of positive PCVAD Scores was 3.9% in generation 26, CG2, and only 1% of these pigs were viremic at 90 days of age. The incidences of PCVAD in generation 25, CG2, and Generation 26, CG1, were greater than 20%; 75% and 93% of the pigs in these respective groups were viremic.

Pigs with PCVAD differed in birth weight, weaning weight, 65-day weight, and 180-day weight, even though some of these weights were recorded well before pigs expressed symptoms of PCVAD. The genetic correlations of PCVAD score with birth and weaning weights were positive, but not significant. However, environmental correlations were negative and significant, indicating that the phenotypic relationship was largely due to environmental effects that reduced early body weights and increased risk of PCVAD.

Mean PCV2 antibody titers in pigs at weaning was high, most likely due to maternal PCV2 antibodies because all these pigs were non-viremic. Antibody titers decreased from weaning to 60 days of age, but 94.6% of pigs were still non-viremic at 60 days of age. Antibody titers continued to decrease from 60 to 90 days of age, suggesting that maternal antibodies were deteriorating. Effects associated with the birth litter were a major source of variation in antibody titers at 60 and 90 days of age, providing further evidence for significant variation associated with maternal antibodies. Other work has shown that high titers of maternal PCV2 antibodies are generally protective against PCV2, whereas low titers are not; however, maternal antibodies do not fully prevent PCV2 infections. Thus, pigs in our study were likely at least partially protected from PCV2 by maternal antibodies to at least 60 days of age.

Implications

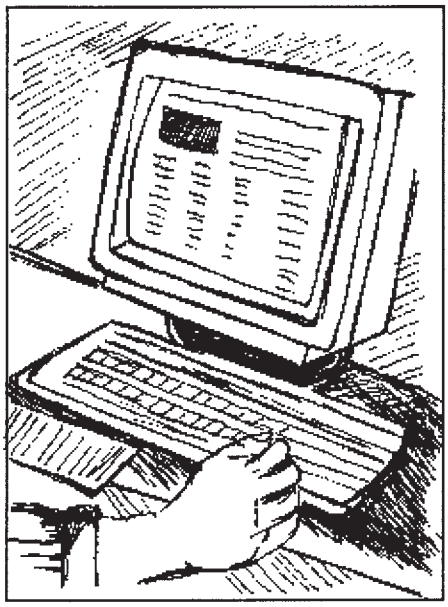
Immune responses to PCV2 are heritable. Thus, genetic selection could be a useful tool to reduce incidence of PCVAD. Even though progress is permanent, several generations of selection will be required to greatly reduce the incidence and this selection must be practiced in nucleus herds and then transmitted through the breeding pyramid to commercial populations. Consequently, it would take considerable time for such a strategy to significantly reduce the incidence of PCVAD in commercial herds. Furthermore, response to selection for PCVAD scores, viremia, and antibody levels will occur only if all pigs in nucleus populations are exposed to PCV2 so that variation reflects genetic variation in the traits. Marker assisted or genomic selection may be more effective as once marker panels with known relationships with response variables are available, selection can be practiced in any population without exposure of pigs to PCV2. Thus genomic selection for resistance to PCV2 and decreased incidence of PCVAD may be the most effective long term selection strategy.

[1]Jared S. Bates was a graduate student, Roman Moreno is a graduate student and research technologist in the Animal Science Department; Alan R. Doster is a professor in the Department of Veterinary and Biomedical Science; and Rodger K. Johnson is a professor in the Animal Science Department.

Explanation Of Statistics Used In This Report

Pigs treated alike vary in performance due to their different genetic makeup and to environmental effect we cannot completely control. When a group of pigs is randomly allotted to treatments it is nearly impossible to get an "equal" group of pigs on each treatment. The natural variability among pigs and the number of pigs per treatment determine the expected variation among treatment groups due to random sampling.

At the end of an experiment, the experimenter must decide whether observed treatment differences are due to "real" effects of the treatments or to random differences due to the sample of pigs assigned to each treatment. Statistics are a tool used to aid in this decision. They are used to calculate the probability that observed differences between treatments were caused by the luck of the draw when pigs were assigned to treatments. The lower this probability, the greater confidence we have that "real" treatment effects exist. In fact when this probability is less than .05 (denoted **P < .05** in the articles), there is less than a 5% chance (less than 1 in 20) that observed treatment differences were due to random sampling. The conclusion then is that the treatment effects are "real" and caused different performance for pigs on each treatment. But bear in mind that if the experimenter obtained this result in each of 100 experiments, 5 differences would be declared to be "real" when they were really due to chance. Sometimes the probability value calculated from a statistical analysis is $P < .01$. Now the chance



that random sampling of pigs caused observed treatment differences is less than 1 in 100. Evidence for real treatment differences is very strong.

It is commonplace to say differences are significant when $P < .05$, and highly significant when $P < .01$. However, P values can range anywhere between 0 and 1. Some researchers say that there is a tendency that real treatment differences exist when the value of P is between .05 and .10. Tendency is used because we are not as confident that differences are real. The chance that random sampling caused the observed differences is between 1 in 10 and 1 in 20.

Sometimes researchers report **standard errors of means** (**SEM**) or **standard errors** (**SE**). These are calculated from the measure of variability and the number of pigs in the treatment. A treatment mean may be given as 11 .8. The 11 is the mean and the .8 is the SEM. The SEM or SE is added and subtracted from the treatment mean to give a range. If the same treatments were applied to an unlimited number of animals the probability is .68 (1 = complete certainty) that their mean would be in this range. In the example the range is 10.2 to 11.8.

Some researchers report **linear** (**L**) and **quadratic** (**Q**) responses to treatments. These effects are tested when the experimenter used increasing increments of a factor as treatments. Examples are increasing amounts of dietary lysine or energy, or increasing ages or weights when measurements are made. The L and Q terms describe the shape of a line drawn to describe treatment means. A straight line is linear and a curved line is quadratic. For example, if finishing pigs were fed diets containing .6, .7, and .8% lysine gained 1.6, 1.8 and 2.0 lb/day, respectively we would describe the response to lysine as linear. In contrast, if the daily gains were 1.6, 1.8, and 1.8 lb/day the response to increasing dietary lysine would be quadratic. Probabilities for tests of these effects have the same interpretation as described above. Probabilities always measure the chance that random sampling caused the observed response. Therefore, if $P < .01$ for the Q effect was found, there is less than a 1 % chance that random differences between pigs on the treatments caused the observed response.

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